TSOMIAN LECTURES

DR SANSOM

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ON THE

TREATMENT OF SOME OF THE FORMS OF VALVULAR DISEASE OF THE HEART

NOTE.

ON the cover of this book is embossed an outline figure of the chest. If a piece of ordinary note-paper be applied to this, and the point of a blacklead pencil be drawn from side to side over the paper, a "rubbing" will be obtained, which will serve as a "chest-chart," and on which the situations of murmurs, the outlines of dulness, &c., can be indicated by coloured marks.

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ON THE

TREATMENT OF SOME OF THE FORMS OF VALVULAR DISEASE OF THE HEART

DELIVERED BEFORE THE MEDICAL SOCIETY OF LONDON

BY

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PREFACE.

THE Medical Society of London has honoured me by expressing the wish that these Lectures should be published. I comply with the request, regretfully conscious that much of imperfection attaches to them.

It was my intention to have included a brief sketch of the treatment of diseases of the aortic valves; I soon found, however, that this, to be consistent with my plan of exposition, was impossible. This plan is mentioned at the commencement of my first lecture; but I may add that I could not approach questions of treatment of any disease without formulating the bases on which I conceive such treatment should rest. To do otherwise would be, in my opinion, to promulgate an unscientific empiricism. In order, therefore, to define the bases for treatment, I have been obliged to touch upon questions of pathology and diagnosis where I have considered that these have not been made sufficiently manifest by extant evidence. As far as possible I have avoided controversy, and have dealt with facts rather than with opinions.

The time allotted to me permitted, for the most part, only a review of the indications for treatment of those diseases of the valves of the heart which are due to the form of endocarditis known as the rheumatic. To have followed my plan of exposition with regard to diseases of the aortic valves, it would have been necessary for me to

reverse the process—to have considered the diseases and disorders of arteries with their effects upon the aorta, and to have differentiated the centripetal or atheromatous from the centrifugal or endocarditic forms of disease involving the aortic valves. It is obvious that this could not be accomplished within the scope of these lectures.

For the material of which I have availed myself I have to thank many kind friends—Dr. T. H. Green, who has permitted me to use some of his drawings; my colleagues at the London Hospital and the North-Eastern Hospital for Children, and the line of indefatigable house-physicians, medical registrars, and clinical clerks at both these institutions.

Lastly, my thanks are due to the indulgent audience that made my task a pleasant one.

84, Harley Street, W.,
May, 1883.

THE LETTSOMIAN LECTURES

ON THE

TREATMENT OF SOME OF THE FORMS OF VALVULAR DISEASE OF THE HEART.

Lecture I.—ENDOCARDITIS.

The Rational Bases of Treatment—Morbid Anatomy—Clinical Investigation—Origin and Progress of Endocarditis—Study of Endocarditis occurring in Children — Pathogenesis — Existing Methods of Treatment—Preventive Treatment.

In my first perplexity as to choice of a subject, when I learned that this Society had done me the high honour of electing me Lettsomian Lecturer for the present year, I thought that I ought to be guided by two considerations. The first, that it behoved me to give of my very best—in other words, that I should address you on a subject upon which I had the most personal experience. Yet I well knew that my best effort would fall far short of my desire and your desert. The second, that, considering the character and traditions of this Society, I should aim at something practically useful. I am very far from decrying the labours of those who pursue Science for her own sake, and I well know that many who have done so have elicited truths which have

eclipsed, in numberless instances, as regards importance and usefulness the results obtained by those who might, from their mode of procedure, be deemed more practical men. But I dared not take a narrow path in mere hope. So I thought it best to review a subject which presents itself very frequently as a therapeutic problem to everyone who is daily occupied in the practice of medicine, and I chose the affections which my title indicates because for many years my thoughts have turned towards them. It seemed to me that it might fulfil a useful purpose if I reviewed our extant knowledge as to the treatment of valvular diseases of the heart, compared these with the results of my own experience, and made, perhaps, a few suggestions as to progress towards precision in the future.

Then as to the point of view whence I could review the subject I felt some doubt. I could proceed from the therapeutic agent to the disease, or from the disease to the agent. Here, with all my difficulty as to how to perform my task, I could not hesitate as to how *not* to do it. I would by no means enunciate a therapeutic dogma, crystallise it into a phrase, and marshal the facts in such wise as they should support it, and if they refused—so much the worse for the facts. Apart from the consideration that such dicta as "*Similia similibus curantur*," "*Contraria contrariis*," etc., present to my mind some of the most pernicious of hasty generalisations of our day, is the one consideration that they are based on the treatment of *symptoms*; and as I shall presently show that the diseases we are about to study are oftentimes accompanied by no symptoms at all, so the practical application of the dogma becomes an impossibility, and its universality an absurdity.

For many reasons I thought it best to consider the phenomena of disease first, and our treatment of them subsequently. My plan then will be to enunciate very briefly the bases on

which I believe our therapeutics ought to rest. These are, in my opinion, (1) the teachings of morbid anatomy; (2) clinical observation of disease-processes and their correlations. Then I propose to review (3) the lessons of the past as to treatment, and (4) to adduce towards the elucidation of the various problems the arguments from analogy afforded by experimental investigation—a mode of inquiry rendered difficult, alas! by the stumbling-blocks which a false sentimentalism has placed in our way.

I. First, then, I will consider *the teaching of morbid anatomy as to lesions of the valves of the heart.* You will understand that I shall do this very briefly, for my object is merely to note them in so far as they may afford a guide to treatment, and when I speak apparently dogmatically I do not make an assertion "*ex cathedrâ*," but in the spirit of an inquirer after truth.

We will first consider the disease which most commonly affects the valvular apparatus of the heart and the adjacent endocardium—the disease known as *Endocarditis.* In briefly reviewing its morbid anatomy much will remain unsaid, but I shall treat it first from the stand-point of mere observation, leaving all speculative questions. I would classify the *first* changes in the endocardium which I shall notice as *exudative.* The curtains or cusps of the valves may be seen to be slightly swollen, and the endocardium to contrast by its dulness with the healthy portions adjacent. The changes are most noticeable at the free edges of the valves, where may be seen isolated or agglomerated bead-like processes. Upon such processes may be observed sometimes little caps of fibrine. The situations where these appearances are most obvious are (1) the line of contact of the aortic cusps at the time of their closure (Fig. 1); (2) the auricular aspect of the mitral orifice (Fig. 2). They are frequently seen also on the surface of the endocardium, in

the neighbourhood of any abnormal thickening of, or growth upon, a valve (Fig. 3), and upon the chordæ tendineæ of the mitral.

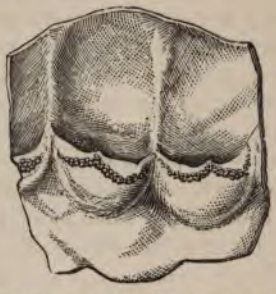

FIG. 1.—Inflammatory granulations on aortic cusps.—(*Dr. Green's "Pathology."*)

A thin section of a valve thus affected is seen under the microscope to differ from healthy valve structure in that its cellular elements are more numerous, and

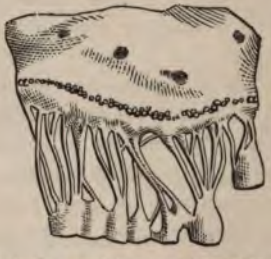

FIG. 2.—Inflammatory granulations on auricular surface of mitral curtains.—(*Dr. Green.*)

especially towards the free edge are closely aggregated. I wish to insist on the fact that, in a valve so affected, even the portions which seem to the naked eye unaffected are really infiltrated with cells. Only the aggregation is

greater at the free edge, and here often the aggregated cells form little very slight concavities on which rest little caps of fibrin. The bead-like eminences observed by the naked eye are, then, according to my view, indications of a more widely spread inflammatory change in the valve than might be at first suspected. In a microscopical section, which displays not only the inflamed valve, but the surrounding structures, one observes that as one

FIG. 3.—Inflammatory granulations in the neighbourhood of a large vegetation depending from an aortic cusp; these granulations probably induced by the mechanical irritation of such vegetation.—(*Dr. Green.*)

approaches the inflamed valve, cells in number are scattered amongst the neighbouring muscular fibrillæ, and near the aortic valves the fibrous structures of the root of the aorta are similarly infiltrated. The fibrous structure of the normal valve is rendered less evident, or is lost. The valve is swollen and infiltrated with cells. This is well shown by the accompanying drawings, taken from specimens prepared by my friend and colleague, Dr. Charlewood Turner. In the case of the healthy valve (Fig. 4) its whole

thickness is shown. The drawing represents a portion of a transverse section of one of the semi-lunar cusps of the aortic valve near its attached border. The upper, or aortic portion, is seen to consist of a narrow band of compact fibrous tissue, the fibrils of which are, for the most part, cut transversely. The lower or ventricular portion is of a looser structure, with its fibres parallel to the plane of

FIG. 4.—Section of Aortic Valve in a state of Health.—(*Hartnack, ob.* 3, *oc.* 3, 72 *diams.*)

section. Small, round nuclei are disseminated through the tissue. The specimen was taken from a well-developed woman, aged twenty-three. In the inflamed valve the increase of bulk is such that the artist cannot represent in reasonable space the whole of the section; the fibrous striation of the deeper portions is not visible, but cells are

abundantly manifest throughout; at the free edge these cellular elements are aggregated at certain spots, and surmounting the margin is a fringe of fibrin. The specimen was obtained from a case of chorea, associated with endocarditis, in a girl, aged sixteen. It is scarcely encroaching on speculation if we conclude that this is

FIG. 5. Section of Aortic Valve in a state of Inflammation, showing the free edge and a portion of the valve-structure.—(*Hartnack, ob.* 3 *oc.* 3, 72 *diams.*)

the recent, the early stage of endocarditis. In looking over the records of sixty-eight post-mortems of cases of valvular disease at the London Hospital, I find that this stage of recent inflammatory change with exudation was observed in nine, *i.e.*, about 13 per cent. The

aortic cusps were affected in five, the mitral in three cases. In one case mitral, aortic, and tricuspid were affected; in another, mitral, aortic, tricuspid, and pulmonic. In one case the tricuspid alone was thus diseased. In such early stage of endocarditis, emboli were noted in two cases—in one in the kidney, in the other in a branch of the pulmonary artery.

The *second form* of endocarditis, or properly speaking valvulitis, to which I shall call attention, is that which I would term the *sclerous* or fibrotic form. Here the valve—and it is the mitral which is affected in by far the greatest frequency—is thickened, but the thickening is not due to swelling of the soft tissue; it is felt to be hard and firm. The endocardium of the auricle near the valve is found to be dense and white. The valve curtains, and often the cords and fleshy columns, are more or less rigid. A patch of the endocardium lining the left ventricle and leading up towards the aortic cusps is sometimes also found white and thick, and the aortic valves themselves may be seen to have undergone similar changes. In this form microscopic section shows that there is a gradual fibrous transformation of the neoplasm resulting in the production of a quasi-cicatricial tissue. In some cases the thickening is such that the structure resembles cartilage—in fact, Dr. Wilks has found well-marked cartilage in such a thickened mitral.* Or, degeneration continuing, calcareous change may take place, and the valve, etc., become of bony hardness. It is evident that this may be considered the chronic form of endocarditis. It was met with in one-fourth of the post-mortems in cases of heart disease which I have mentioned. The effects produced upon the mitral and aortic orifices will be treated of in future lectures. In this class of cases vegetations were observed in the proportion of seven instances

* "Pathological Anatomy," by Wilks and Moxon. Second edition, page 134.

in twenty cases—on the mitral and adjacent auricle in three cases, the aortic in two, the tricuspid one, the aortic, mitral, and tricuspid as well as in the auricle in one case. Infarcts were noticed in branches of the pulmonary artery (five cases), spleen (five), kidneys (two), brain (one), retinal artery (one), intestines (one).

A *third form* of endocarditis, which I think of practical importance to distinguish, is that which is *secondary to endarteritis* (atheroma). In this form it is the aortic valves which are affected in a large majority of instances. Patches of soft, flabby swelling may be seen in the lining membrane of the aorta close to the aortic cusps, involving them in the change, and perhaps causing the inversion of one or more. Or yellowish patches may be observed, in some cases covered by a soft pulpy material, the blood perhaps forcing its way at some softened spot between or within the arterial coats. Or the root of the aorta may be hard and thick, the thickening being of cartilaginous consistence, and in such thickening the cusps of the valve may be involved. Or in like situation and with like deformity of valves there may be a bony or stony hardness—a calcareous change. The evidence obtained by microscopic investigation is to the effect that in the swollen soft patches are abundant exudation-cells with hyaline or slightly fibrillar matrix. These occur mostly as swellings of the internal coat; but Dr. Wilks has observed them in all the coats of the vessel. The yellow patches show fat granules, and sometimes cholesterine crystals. There is evidently a fatty degeneration of the inflammatory neoplasm. In the fibrous or semi-cartilaginous variety there is more fibrillation and fewer cells; and in the hard and bony form there is a deposit of earthy salts in the interstices of the fibrous tissue. In this category came twenty-seven of the sixty-eight autopsies of heart disease which I have recorded. What I may term the soft stage

was observed in eight instances, fibrous thickening in seven, calcareous change in five. The mitral valve was also thickened or atheromatous in seven cases, the tricuspid in two. In one case where there was calcareous transformation, ulceration of one cusp of the aortic valve was also observed. Infarcts were discovered in three cases—less commonly, it will be observed, than in the other forms of valvulitis—in the kidney in one case, in spleen and kidneys in another, and in middle cerebral artery in a third.

The *fourth* and last form of endocarditis, as demonstrated by post-mortem examination, to which I shall call attention is that termed *ulcerative endocarditis*. Swollen and dull portions of the endocardium of the valve may be seen to present here and there a yellowish or greyish discoloration, and to be covered by a finely granular *débris*. The superficial endocardium in such situations has become necrosed. Through such breach blood may find its way, and, spreading between the layers constituting the valve, may form an aneurism thereof; or, the ulceration extending through both layers, the valve may be perforated. More commonly a considerable portion of the valve is eroded, and upon the eroded surface fibrine is deposited in the form of single or multiple vegetations. The finger readily detaches these vegetations, and the surface below them is found to be covered by a friable material. Microscopic examination has demonstrated in a very large number of cases the presence on the ulcerated surface and in the tissue of the valve of aggregations of micrococci. The accompanying engravings (Figs. 6 and 7), from preparations made by my colleague, Dr. Stephen Mackenzie, show the presence and positions of colonies of micrococci in ulcerative endocarditis, and the appearances of some of the detached masses (zoogloea). In this form of valvulitis *embolism is* the rule, and such emboli are sometimes *infec-*

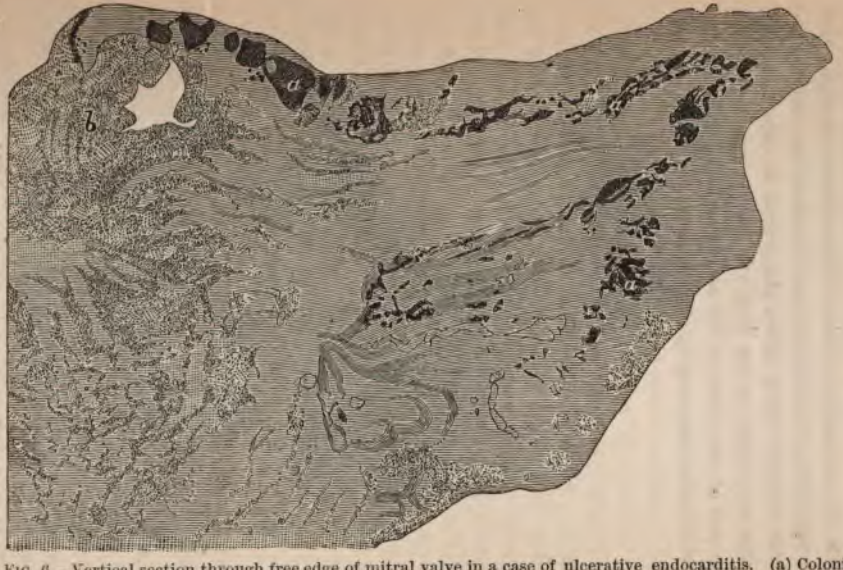

FIG. 6.—Vertical section through free edge of mitral valve in a case of ulcerative endocarditis. (a) Colonies of micrococci; (b) Corpuscular infiltration (inflammatory). *Moderately magnified.*—(*English, 2 inch o.g. 14 diams.*)

for a *second* attack of rheumatic fever the proportion was 48·5 per cent. In those who had suffered from two or more previous attacks the proportion was 59·0 per cent. In 1881, the valvular morbility was in the first attack slightly reduced (viz., 41·8 per cent.), whilst in the second attack it had greatly risen (viz., to 70·8 per cent.), and after two or more attacks stood at about the same ratio (67·1 per cent.) In 1882 valvular changes were noted in 48·1 per cent. of the patients suffering from a first attack; in 66·6 per cent. of those suffering from a second attack; and in 77 per cent. of those suffering a third attack and over.

To modify the plan of observation, the heart was *noted as healthy* in 1880, in cases of a first attack of rheumatic fever in 50 per cent., in cases of a second attack 40 per cent., and after two or more attacks 20 per cent. In 1881, the record of "healthy heart" was in the ratio of 37·1, 19, and 22·8 per cent. in the three classes respectively. In 1882 the heart was healthy in 46·8 per cent. of those admitted for a first attack; in 25·2 of those admitted for a second attack; and in 18·7 per cent. of those who had, previously to admission, suffered two or more attacks.

We may now inquire by what signs the advent of endocarditis in the course of acute rheumatism is declared? I exclude those cases which are complicated by pericarditis, because those are out of the scope of my subject. First, as regards *symptoms*. These, according to the experience of many with which my own observations are entirely in accord, are by no means characteristic. Oftentimes there is absolutely no subjective sign which might give rise to the suspicion that the lining membrane of the heart is becoming involved in a serious disease. The course of the rheumatic fever appears to be modified in no appreciable degree. I am aware that some observers have laid greater stress on the prevalence of such subjective signs. The late Dr.

Sibson, for instance, states that in nearly every one of his cases developing heart complications in acute rheumatism the inflammation "pronounced itself by the immediate language of the heart itself, by pain in its region, by the anxious expression of the face and its dusky or glazed hue, and by the disturbed breathing."*

Next, as to the *physical signs* by which the endocardial implication is indicated or rendered probable. I believe the most frequent sign to be a *prolongation of the first sound of the heart*. Sir William Gull and Dr. Sutton have noted this sign. They say: "Such a prolonged first sound not unfrequently in the course of a few days becomes a well-marked mitral bruit. . . . It also occasionally happens that the first sound is prolonged at the apex, and continues so until the patient is almost, if not actually, convalescent; and then this prolonged sound becomes a decided mitral murmur."† Dr. Sibson made a similar observation. Prolongation of the first sound was noted by him in eighteen out of twenty-two cases of threatened rheumatic endocarditis.‡ My own view as to the significance of this sign is that it is due to an impairment of the valvular element of the first sound. The curtains of the valve being swollen, the flap of their closure is rendered less manifest; the ear consequently perceives, for the most part, the muscular element of the systolic sound. The period of the disease at which manifestation of the involvement of the endocardium occurs is an important, though a debateable, question. Hayden placed it from the sixth to the ninth day of an attack of acute rheumatism;§ Fuller from the sixth to the twentieth day. Gull and

* "Address in Medicine," *British Medical Journal*, August 13, 1870, page 161.

† *Medico-Chirurgical Transactions*, 1869, page 82.

‡ *Loc. cit.*, page 162.

§ "Diseases of Heart and Aorta," page 799.

Sutton say, however: "Experience teaches that the heart becomes diseased at the very outset of the rheumatic fever, before the patients enter the hospital,"* and Sibson was in accord with this observation: "The prolongation of the first sound, when present, was generally audible on the first day." I consider that though the prolongation or murmurish character of the first sound may be heard at variable periods of the evolution of rheumatic fever, it is very common to find it, as the observers last quoted have said, at the very earliest periods of evolution of the disease. I shall again call attention to the importance of this observation. But even though a distinct mitral murmur be noted this must not be taken as conclusive evidence of disease of the valves, for it may be due to regurgitation from passive yielding of the ventricular muscle. This we shall consider in the lecture on Mitral Regurgitation.

A prolongation of the first sound or the production of a veritable systolic murmur does not, however, constitute the only sign of involvement of the endocardium in disease. To one sign I wish to call particular attention, chiefly because I want more evidence on the point. I have observed, as an early sign, *reduplication of the first or of the second sound* of the heart, and, so far as my experience has gone, when I have observed this sign the resulting change upon the valve has induced, not mitral regurgitation, but mitral stenosis. I have formerly before this Society developed my views as to the manner in which such reduplication is effected.† And, again, the change may be noted exceptionally in the aortic and not in the mitral valve. I have observed, during the evolution of rheumatic fever, a musical

* *Medico-Chirurgical Transactions, loc. cit.*, page 80; *British Medical Journal, loc. cit.*, page 162.

† *Proceedings of the Medical Society of London*, vol. v.

diastolic murmur become manifest at the base of the heart. My view is that such murmur is caused by the vibration of a pedunculated vegetation depending from an aortic cusp.

The next inquiry I would make is, Whether is there any causal relation between the pyrexia of rheumatic fever and the occurrence of endocarditis? Wunderlich says: "Cardiac complications are by no means excluded by the absence of fever."* My own experience is entirely in accord with this statement. Again, cases of rheumatic fever which manifest hyperpyretic temperatures are not accompanied by an abnormal proportion of valvular complications. In fact, the report of the Committee of the Clinical Society on Hyperpyrexia in Acute Rheumatism, states that endocarditis was a little less frequent in such cases than in rheumatic fever generally.† The conclusion, therefore, is irresistible, that there is no relation of causation between pyrexia and endocarditis.

Some authors have considered that there is a relation between the severity of an attack of rheumatism, the extent of the polyarthritis, and the development of valvular disease. I can only say that such is not my experience. This will engage our attention immediately.

Let us now inquire concerning those cases of endocarditis which are not associated with a history of acute or subacute rheumatism. These may conveniently, for purposes of investigation, be divided into two classes—(1) those which are observed in early life; (2) those which develop after maturity. In the latter class are those cases of gradual onset which involve the aortic orifice, and sometimes the mitral, which are traceable to subinflammatory changes at

* "Medical Thermometry," New Sydenham Society's translation, page 390.

† *British Medical Journal*, June 3, 1882, page 807.

the root of the aorta, and degeneration subsequently. In these cases the endocarditis and valvulitis are consecutive —they have no necessary connexion with rheumatism, and their consideration may be conveniently deferred.

The study of endocarditis as it occurs in the early periods of life is, however, at the point at which our investigation has hitherto been advanced, a matter of very great importance. Almost every practitioner is familiar with the fact that cases of disease of the valves present themselves which have shown evidence of such disease for many years, from very early periods of the life of the patient; and yet inquiry fails to elicit that the subject of such disease has ever suffered from rheumatism in any form. It is surely a matter of importance, therefore, that we should endeavour to learn how such disease originates in the period of childhood.

I have elsewhere discussed this question at some length,* and I shall here only call your attention to a summary of such points as I think are absolutely necessary to bear in mind when we are considering endocarditis with a view to treatment. I may, however, cite some evidence supplementary to my former lectures, derived from a summary of more recent cases prepared for me by Mr. J. A. West, our House-Surgeon, and formerly our Registrar at the North-Eastern Hospital for Children.

In acute and subacute rheumatism in the child, it has been considered by West, Rilliet, Barthez, and others that the proneness to endocarditis is greater than in the adult. Rosenstein† has combated this view; he considers "the disposition to endocardial affections on the whole smaller in childhood than after puberty." My experience

* "Clinical Lectures on Diseases of the Heart in Children," *Medical Times and Gazette*, 1879.

† "Ziemssen's Cyclopædia," vol. vi., page 85.

entirely coincides with that of Dr. West, and is against Rosenstein, whose only recorded argument is that "he has repeatedly seen cases of rheumatic arthritis, even in children, which were not followed by endocarditis." Typical rheumatic fever is much less common in the child than in the adult; the articular manifestations are slighter, but I consider the morbility of the endocardium to be greater. Of thirty-two cases of acute and subacute rheumatism occurring in children under twelve years of age admitted into the North-Eastern Hospital during the past three years, twenty, or 62 per cent., presented signs of endocardial affection. The development of endocarditis, however, in the child has not so close a relation with the other phenomena of rheumatic fever as in the adult. It may precede, or may succeed, even after long periods, the attack. We meet with cases of endocarditis in children by no means uncommonly where the manifestations of rheumatism are very slight. There may be no history of subacute rheumatism, but only very slight pains, often designated "growing pains." Valvular complications have been noted by me in the following proportions in the three classes of cases: (*a*) acute rheumatism, forty-seven cases; (*b*) subacute rheumatism, twenty-one cases; (*c*) rheumatoid pain, eight cases.

Again, the manifestation, or, rather, indication, of rheumatism may be even slighter still. I have noted endocarditis in cases where *eruptions* have been the only indications (if so they be admitted) of the rheumatic diathesis. Such eruptions are *eczema*, *erythema* (*e. circinatum* or *e. marginatum*), and *purpura*.*

But there are other diseases besides rheumatism in the child with which endocarditis stands in close relation. These are

* *Vide* "Lectures on Diseases of the Heart in Children," *Medical Times and Gazette*, December 27, 1879, page 711.

chiefly scarlatina and measles. In relation with scarlatina, endocarditis may occur either with or without the intervention of articular symptoms. Post-scarlatinal rheumatism is well known, and bears a close similarity to ordinary rheumatic fever; associated endocarditis is therefore rendered probable. But I have shown from recorded cases that such endocarditis may become manifest after scarlatina, not only without the intervention of articular phenomena, but long after the period of fever has passed, and during a time when there is no elevation of the temperature of the body, no pyrexia whatever.*

Again, there is evident proof that endocarditis can arise in close relation with *measles*. I have recorded a case in which both pericarditis and endocarditis occurred a fortnight after the commencement of convalescence from measles. At this time a perilous attack of chorea developed. There was here no obvious manifestation of rheumatism, nor hereditary tendency thereto. It appears to me that the influence of measles in predisposing to endocarditis has been much under-rated. And, *à fortiori*, the frequent sequence of these diseases, as observed in children, becomes an agency, as I think very probable, not only to the production of the endocardial disease, but to acute rheumatism itself. To take examples—

1. Scarlet fever, measles, and subacute rheumatism in one year; mitral regurgitation.

2. Scarlet fever at age of two; second attack at eight, followed by measles and rheumatoid pains; mitral regurgitation.

3. Measles at age of two, scarlet fever at three; mitral regurgitation and aortic obstruction.

* "Lectures on Diseases of the Heart in Childhood," *Medical Times and Gazette*, October 25, 1879, page 472.

Of nine other cases in which measles was noted in the previous history of cases manifesting endocardial murmurs, acute and sub-acute rheumatism were manifested in four.

After measles, just as after scarlatina, endocarditis, or pericarditis, or both combined, may develop, with no signs of pyrexia.

Excluding all these probable causes, however, there yet remains a very considerable minority of cases of endocarditis in children in whom no traceable disease has led up to the valve-deterioration. The condition is only betrayed by various morbid conditions, the results or concomitants of the valvular disease. I have noted twenty-seven of such cases. They have been marked by (*a*) disorders of the nervous system — hemiplegia, hemianæsthesia, epilepsy, chorea ; (*b*) disorders of nutrition—wasting, anæmia, etc. ; (*c*) disorders of respiration or circulation—cough, dyspnœa, or the usual phenomena of progressive cardiac failure.

Sufficient is this evidence to prove, I think, that in the child endocarditis can arise and progress without special symptoms, without pyrexia, without the disturbing influence of any acute disease. It may be asked, however, whether the form of endocarditis in such cases differs in any way from that which we know as the rheumatic form. The answer is given by the post-mortem evidence. There is no obvious difference from the essential features of rheumatic endocarditis, such as we find in the undoubtedly rheumatic subjects.

Such is the evidence—the important evidence, as I estimate it—to be obtained as to the rise and progress of endocarditis from the clinical observation of the cases occurring in children. It becomes us, however, to revert to the general subject, to the disease as it is seen in adults, and to inquire whether there are any other diseases with which we

find endocarditis associated. In many acute fevers, typhoid for example, it is so rare that I consider it most probable that some other factor must have been in existence in the cases in which it has been observed. In *diphtheria* it has been said to be frequent. M. Labadie Lagrave has described it as occurring in fifty cases out of a hundred. In this country, however, observers have, so far as I am able to ascertain, noticed no such association. Certainly I have not myself, and I cannot refrain from concluding that special features (might it have been a relation with scarlatina?) marked the epidemic, the cases of which M. Labadie Lagrave has so carefully recorded.* I cannot help thinking that the term *diphtheritic* as applied to *ulcerative* or *necrosing* endocarditis has been productive of confusion.

An association between septicæmia and endocarditis has been noted, and also, as my own experience has confirmed, with puerperal conditions. M. Lancereaux recorded five cases in which it was thus observed. "In all these cases," he says, "the valvular affection manifests itself with characters of striking similarity; localisation to one portion only of the diseased orifice, exuberant vegetation, termination by necrosis, and ultimate phenomena of infection."† In fact, this is a variety of ulcerative endocarditis.

And now let us approach the question of the *pathogenesis* of endocarditis. Excluding the ulcerative form, which we can more conveniently discuss subsequently, and the form which is secondary to arterial degeneration (because the endocarditis is here due to the involvement of adjacent

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* "Des Complications Cardiaques du Croup et de la Diphthérie." Paris, 1873. *Cf.* Morell Mackenzie, "Diphtheria," page 54. London: J. and A. Churchill. 1879.

† "Anatomie Pathologique," page 537. Paris: Victor Masson et fils. 1871.

endocardium in the subinflammatory changes which are an essential feature in the aortic disease), there remains the large class which includes the exudative and the sclerous forms that I have described. These are, I consider, from their clinical and pathological characters, to be grouped together as the *rheumatic form* of endocarditis — the sclerous being the chronic form of the exudative.

Now, I think that our clinical experience has taught us that such endocarditis may arise in an extremely insidious manner, that it may give no evidence of its rise and progress by signs nor symptoms, nor even by rise of temperature. It may probably be in existence at the very earliest period of an attack of rheumatic fever, and even with great probability precede it unnoticed (for such may be the significance of the muffled or prolonged first sound heard at the earliest period of such disease). Moreover, it may progress after the attack of rheumatic fever, causing gradual induration or retraction of the valve in a patient who may have been discharged from treatment as free from cardiac complication. Such is the disease—insidious in onset, course, and character—the causes of which we have to consider.

However occult its origin and course, we must allow that the inflammation of the endocardium is an integral part of the rheumatic process, that it is produced by the same agency which in many cases, though not in all, produces inflammation of the fibrous textures of the joints. Such morbid agent is, without doubt, distributed by the blood. The question occurs, Is it introduced from without or developed within? The view of its extrinsic nature has been forcibly argued lately, especially by Dr. Maclagan.* The hypothesis is that the *materies morbi* which produces it is a

* "Rheumatism: its Nature, its Pathology, its Successful Treatment." London: Pickering and Co. 1881.

form of malaria, and, as such, is of the nature of a living, probably a fungoid, organism. Against such a view is, I think, the point which I have brought forward as so strongly borne out by observation—that endocarditis is not a pyrexial disease. Our knowledge of the action of low organised forms upon the living body tells that they give rise to fever. It is, to my mind, highly improbable that proliferating germs introduced into the blood should cause an inflammation of the endocardium, and yet fail to increase the general temperature of the blood. Moreover, the hypothesis fails to explain, in my opinion, many of the phenomena which are explained by the older view that the origin of the *materies morbi* is from within.

Let us consider, therefore, the second proposition, viz., that the endocarditis as well as the other phenomena of rheumatism are due to a *perverted retrograde metamorphosis.* The evidence seems to me to point to this theory as the true one. First we have hereditary proclivity. This, I consider, obtains, not only as regards rheumatism in general, but endocarditis in particular. Secondly, proximate causes; those which I consider proven are sudden exposure to cold, the influence of certain acute specific diseases. What characterises those acute specific diseases, scarlatina and measles? An implication of the skin in a morbid process in both, of skin and kidneys in one. All such causes have one character in common—production of an impediment to elimination. In a large number of instances I agree, of course, that proximate causes are not in evidence. In the next place, the proved conditions of the disease. An extremely acid sweat is excreted; lactic acid has been demonstrated therein; but it is not proven that lactic is the *only* acid thus excreted. Certainly there must be some other agent to communicate the peculiar odour which the

perspiration manifests. Again, the urine is abnormally acid. And the saliva, which is normally alkaline, is, in rheumatic conditions, decidedly acid. Furthermore, the blood is abnormal in that it contains an undue proportion of an excrementitious product, viz., fibrin; it is highly coagulable. What is not proven, however, is that the blood is *acid;* on the contrary, in rheumatism the blood-serum is alkaline.* At this step of the inquiry, experimental evidence comes to aid us. Dr. B. W. Richardson, to test the old theory of Prout, that lactic acid is the pathogenetic agent, injected a solution of lactic acid into the peritoneal cavities of animals, and found afterwards undoubted evidence of the production of recent endocarditis in the valves of the *right side* of the heart. These observations have been confirmed by Rauch, but the conclusion that the lactic acid is the *vera causa* of the inflammation has been contested by Reyher on the ground that endocarditis is common in dogs as an idiopathic disease. I think we may readily dismiss such an objection, from the fact that Dr. Richardson's observations give convincing proof of a *recent* inflammation as from an irritant cause, and they have been confirmed by evidence, which, though clinical, is also experimental. Dr. Balthazar Foster has shown that in one case, after ingestion of lactic acid in the human subject, phenomena in all respects corresponding with those of acute articular rheumatism were produced; "they came on when the acid was taken, and ceased when it was discontinued."†

This evidence, I take it, valuable as it is, is illustrative and analogical rather than direct and dogmatic. For me, at least, it shows that lactic acid can be *an agent* in the

* *Cf.* Charcot, "Lectures on Senile Diseases," Sydenham Society's translation, page 162.

† *British Medical Journal*, December 23, 1871, page 722; and "Clinical Medicine," page 152 (London: Churchill, 1874).

production alike of rheumatism and of endocarditis; but it does not prove that it is the *only* agent. We have no proof that the free acid exists in the blood in the disease, but yet it seems to me that we have full and sufficient proof that the normal retrograde metamorphosis is greatly altered; that the blood is changed; that in the course of metabolism many products are formed, with the result that lactic and other acids are excreted in abundance. Sufficient, this, I think, for our present purpose. Is it not probable that the pathogenetic agents are many—I mean, that there are numerous products between fibrin on the one hand, and the excreted morbid acid on the other, capable of giving the irritating impulse?

One step further in this inquiry. The *rôle* of the nervous system in this connexion may be a very important one, and we may ask whether there may not be a portion of the central nervous system specially concerned in the control of the chemical processes of metabolism, just as there is probably a centre which regulates the temperature of the body. Dr. P. W. Latham has advanced the theory that there is such a centre—that such a centre may be disturbed by external cold or by the accumulation of lactic acid in the blood. So he considers that the phenomena of rheumatism may be induced by an intra-spinal change, just as the arthropathies are induced in locomotor ataxy. And if the disturbance of such centre involve also the neighbourhood of origin of the vagus, cardiac, pulmonary, or pleuritic complications may be developed.*

I must now approach another part of my subject, and inquire concerning the efficacy of extant methods of treat-

* "Some Points in the Pathology and Treatment of Acute Rheumatism and Diabetes," *Lancet*, January 8, 1881, and *British Medical Journal*, January 14, 1882.

ment in regard to rheumatic endocarditis. It has been claimed of almost all methods of treatment of rheumatism that have been advocated that they have been instrumental in controlling or preventing the cardiac complications of the disease. The individual experience of observers has been cited again and again to point the efficacy of this or that remedy or method in mitigating the chief danger of rheumatic fever. Yet proof of such vaunted efficacy has soon been found to be unsatisfactory, and it may be confidently asserted that no antidotal treatment is yet known—that we have, for instance, no drug which can influence endocarditis as quinine influences ague, or as mercury and iodide of potassium influence syphilis. The discussion, so ably sustained in this Society during the last session, which has been fully reported, has put the claims of various forms of treatment of rheumatic fever to a numerical test.

The results of treatment by rest and mint-water, by alkalies, by blistering, and by administration of salicin and its compounds, were compared, and it is fair to assume that if any agent other than these had been efficient in the treatment of rheumatic fever or of endocarditis, evidence would have made this apparent. The result of the discussion, which it is unnecessary to epitomise,* was to show a strong concurrence of testimony to the effect that the administration of salicin or the salicylates decidedly reduced the suffering and the fever of rheumatism, but in no marked degree influenced the development of endocarditis and other cardiac complications. *Primâ facie* this seems to be a strange conclusion, for one might imagine that an agent that subdued so decidedly the pain and fever which must contribute to disturb the heart, even if it had

(*) *Vide Lancet* of December 17, 24, and 31, 1881, January 7 and 28, 1882.

no pronounced effect upon the rheumatic process within the heart, would, with great probability, influence for good the inflammatory process in pericardium as well as endocardium. The conclusion is forced home, however, alike by individual experience—for we find that pericarditis and endocarditis are shown by physical signs to arise and progress in patients who are fully under the salicin treatment—and by statistical inquiry from large numbers of cases treated by the salicin compounds compared with those treated in the pre-salicylic era, such as has been carefully followed out by Dr. Gilbart Smith.* Dr. Maclagan, to whom the profession and the public are indebted for the introduction of agents which have, at any rate, been proved to contribute to the comfort of suffering patients, himself allows that the hopes that they would ward off cardiac complications have not been realised.† He considers the reasons for such failure to be —(1) that endocarditis has often begun in an attack of rheumatism before the sufferings of the patient have been so pronounced as to call for treatment; (2) the inflamed endocardium can never, from the incessant motion of the heart, be placed in the conditions of rest which are necessary for cure. I endorse both these propositions, and will add to them.

To put the matter clinically or practically. We observe, let us assume, a patient in a first attack of rheumatic fever. He presents (A) a murmur indicating an endocardial complication. I think I must have convinced you that such endocarditis may have arisen not during the attack from which he is at present suffering, but from the disease acquired insidiously at some time previously. It is obvious that any

* *Lancet*, January 28, 1882, page 135.
† "Rheumatism," page 266. Pickering.

remedy would fail to influence the cardiac complication in such a class of cases. Or, (B) a modification of sounds or actual systolic murmur developing at the apex makes us suspect the present rise and progress of endocardial inflammation. But such may have had its commencement long before the advent of the other symptoms, for no sign will betray the gradual swelling of a valve. A swollen valve is not necessarily incompetent. On the other hand, a veritable systolic murmur at the apex is no conclusive proof of endocarditis, for it may be due to adynamia of cardiac muscle. Here, then, is a double source of fallacy in the statistics of the cardiac complications of rheumatism. Or, (C) the patient manifesting no evidence of valvular impairment is at the termination of his attack of rheumatic fever discharged as free from cardiac trouble. Sir W. Gull and Dr. Sutton have said that "if the patients pass the first few days of the rheumatic fever without the heart becoming involved, then they do not contract heart disease during the later part of the rheumatic attack."* Is such a conclusion justified? I think not. A valve may be inflamed and give no evidence of incompetence; the patient may be discharged and show no signs of cardiac trouble, but a slow process of shrinking or sclerosis may be going on, and when the patient next presents himself there may be undoubted evidence of endocardial mischief. This is, I consider, by no means of infrequent occurrence, and is one reason why a second attack of rheumatic fever is attended with such notable numerical evidence of an increased ratio of cardiac complications.

For such reasons as these I think it impossible, the sources of error being so numerous, that we can get from statistical inquiry satisfactory evidence as to the efficacy of different plans of treatment in warding off endocardial

* *Medico-Chirurgical Transactions*, vol. lii., page 80.

disease, and I dissent from those who hold that a remedy which is efficacious in the treatment of acute rheumatism ought to show, on numerical inquiry, a favourable influence on the correlated heart disease. I consider the treatment by salicin and the salicylates, even though no good results are manifest as regards cardiac complications, to be the most favourable to the patient of all forms of treatment hitherto known.

In such case it may be legitimately asked whether I adopt an altogether pessimist view of the treatment of endocarditis. Can nothing be done? My answer is—Much, but it must be in the direction of *preventive treatment*.

My own experience is strongly towards the conclusion that endocarditis is more prevalent, as well as more extensive and severe, among the poor than among the well-to-do. This question is one that might with advantage be put to the numerical test; we greatly want the evidence of the family practitioner to compare with that afforded by our hospital statistics. The predisposing causes to the advent of endocarditis, which, as I have shown, can arise without the intervention of obviously rheumatic phenomena, are most probably—(1) exposure to vicissitudes of temperature; (2) an irregular and improper dietary. These are the impulses to a perverted nutrition, resulting in the retention within the blood of those excrementitious products which we may call "the rheumatic poison." Attention to the clothing and proper feeding of infants and children constitutes, in my mind, therefore, the treatment of first importance as regards endocarditis. There is no need nowadays to insist on the value of preventive treatment as regards the zymotic diseases. This is well recognised. Is it not quite as important as regards the subtle disease we are considering? I would, whilst recognising the difficulties of such proceeding,

strongly recommend the periodic medical examination of children, even though they present no obvious signs of disease.

Of no less importance is the treatment in regard to the zymotic diseases which are correlated with endocarditis, viz., scarlatina and measles. The subject of an attack of scarlatina should be watched with great care for long periods after convalescence. Moreover, the slightest sign of throat-ailment, especially with children, should be looked upon with suspicion. I have no doubt whatever that in a large number of instances ulcerative tonsillitis of zymotic type occurs in children unnoticed and unknown, and that in many such a renal complication is instituted which is also neglected. The rise of endocarditis in such a case is, as I have said, not during the period of fever. I do not recognise the influence of morbid germs in *directly* occasioning the inflammatory change in the valves; but subsequently, such may be developed even after long periods. The teaching I would enforce, therefore, is that the subject of scarlatina or of the allied forms of throat-affection should be watched, protected, dieted, and treated for periods much longer than is now usual. And as regards measles, there is, unfortunately, a widely spread tendency to regard this as a very slight ailment that requires little or no treatment. Experience teaches, however, that it is not only the immediate precursor of broncho-pneumonia frequently, and heart disease occasionally, but that it effects a deleterious change upon the powers of nutrition, which lasts, as in the case of scarlatina, for long periods. The subsequent treatment, therefore, of the subjects of measles should, in my opinion, be much more protracted than it is at present.

Such is an outline of what I consider the common-sense treatment of the first causes of endocarditis. During its

rise and progress in an attack of rheumatism, I prefer the treatment by salicin or the salicylates in sufficient doses (usually gr. xx. every two, three, or four hours, till subsidence of the pain and pyrexia, and afterwards the same dose thrice or twice a day). From the evidence of Dr. Isambard Owen there is a good case in favour of combining with this the administration of full doses of alkalies.* Vesication by application of liq. vesicatorius in the left axilla I think also of service.

It now only remains for me to allude to the clinical significance of *ulcerative endocarditis* with regard to indications for treatment. It happens sometimes that this affection arises and runs its course with little or no evidence that the endocardium is affected. Such cases often present a strong resemblance to typhoid fever. Hence treatment is of no avail—the disease is uniformly fatal. By far the most frequently the disease is engrafted, as it were, on chronic disease of the valves. It appears to me that such cases can be divided into two classes—the infective and the non-infective. In the infective cases there are extraordinary disturbances of temperature, multiple emboli, septicæmic signs, or even abscesses. It is in such that micrococci are discovered. I believe them to be associated with some subtle zymotic influence, or a virus, as in the puerperal cases. It is not that the micrococci induce the endocarditis, but they complicate the already existent endocarditis by bringing about necrosis of the diseased tissue. In other cases, though nearly all are characterised by embolism, the proof of infection and, as I think, the probabilities thereof are wanting. In a case lately under my care in the London Hospital there was no marked pyrexia whatever, the temperature

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* *Lancet*, January 28, 1882.

never exceeding 101° Fahr., and for the most part keeping close to the normal. I consider it most probable that in some such cases the ulceration is induced by mechanical causes. Drs. Wilks and Moxon have pointed out that a great mass of vegetation may cause ulceration of the heart-wall by direct pressure or a fibrinous clot swinging in the blood-current, and may, coming sharply into contact with the muscle, so by friction start an ulcer.* In like manner, I think it very probable that a weighty vegetation or mass of vegetations upon a valve may, by agitation in the blood-current, so disturb the nutrition of the endocardium which constitutes its base as to start the process of necrosis.

The treatment of ulcerative endocarditis when once established is hopeless, but the lessons taught by a study of the cases are, I consider—1. That more than ordinary care should be exercised to keep the subjects of valvular disease of the heart from possible sources of infection; 2. That any threatening of endocarditis should be treated by the most perfect physiological rest attainable; 3. That nutrition should be sustained to the highest degree practicable.

* "Pathological Anatomy," second edition, page 129.

Lecture II.—MITRAL REGURGITATION.

Morbid Anatomy—Mitral Regurgitation in Anæmia, in Neuroses of the Sympathetic, in Acute Fevers, in Rheumatism, and in Conditions of High Arterial Tension—Treatment to Restore Compensation—Digitalis—Belladonna—Casca—Caffeine—Convallaria Maïalis—Morphia—Alkalies, etc.

I have to ask your attention to the subject of the treatment of various conditions of disease associated with a certain imperfection in the mechanism of the heart—an imperfection of closure of the left auriculo-ventricular orifice at the time of systole, occasioning the reflux of a portion of the contents of the left ventricle into the left auricle, the mitral valve being inadequate to close the orifice.

Pathological anatomy teaches that such result may be brought about by several varieties of morbid change:—

1. By dilatation of the left ventricle without structural disease of the valve. So the free borders of the curtains are drawn upon by their circumferential attachments, and prevented from a perfect apposition in systole.

2. By the changes in the valve-curtains, the tendinous cords and fleshy columns induced by endocarditis, and the process consecutive thereto. Vegetations about the orifice may prevent its perfect closure. Or the valve being thickened, its segments may be imperfectly coäpted. Or curtains, cords, and columns, any or all, may become shrunken, thickened, fibrous, or cartilaginous from sclerous

change. Or from deposit of earthy salts, the valve and orifice may be hard and calcareous.

3. The valve-curtains, cords, or columns may become ruptured, and therefore incompetent. It has been supposed that this may occur from sudden strain in a healthy heart; but Drs. Wilks and Moxon have given strong reasons for the conclusion that there must have been some dilatation, at least, of the left ventricle previously. They consider that this accident is not of infrequent occurrence, and say—"The snapping of an overstrained mitral tendon in a dilated heart we believe to be a relatively very common cause of severe heart-disease, converting the very bearable trouble of a moderate dilatation into a hopeless disablement."*

4. Patches of atheromatous disease may be observed upon the valve, with consecutive degenerative change, rendering it inadequate.

5. Portions of the valve and the surrounding structures may be destroyed by ulceration.

Such are, in brief, the changes which are observed on post-mortem examination to render perfect closure of the left auriculo-ventricular aperture impossible.

Mitral regurgitation is, however, not to be wholly interpreted by pathological anatomy. It is to clinical investigation that we must chiefly look for guidance. We ask ourselves, first, from what sign observed in the living body do we infer that the mitral orifice is incompletely closed in systole? The answer is, that there is a *consensus* amongst observers that a murmur heard with the first sound at the apex of the heart, localised at this point, conducted externally towards the left axilla, or to the back in the neighbourhood of the angle of the left scapula, indicates that there is in existence

* *Cf.* "Pathological Anatomy," by Drs. Wilks and Moxon. Second edition. London: John Churchill and Sons. 1875.

a condition permitting regurgitation into the left auricle. The sign is almost, though not quite, pathognomonic. The only condition with which it is likely to be confounded is, in my opinion, pericardial roughening at or about the apex. I have never known a difficulty about the differential diagnosis in the case of adults, but I have observed such difficulty several times in children. In cases of children I have repeatedly said that the quality, character, and situation of a systolic apical murmur will not declare with precision whether there is exocardial or endocardial disease. My House-Physicians at the North-Eastern Hospital for Children have observed this with me. A murmur which has been ascribed to mitral regurgitation by competent observers has been proved on post-mortem examination to be due to roughening of or fibrinous exudation on the pericardium in the neighbourhood of the heart's apex.

The difficulty of diagnosis is, however, an infrequent one, and we may conclude that in a vast majority of cases the existence of a murmur having the characters which I have mentioned, indicates a condition of mitral regurgitation.

Combined clinical and necroscopic observation, however, soon convinces us that in certain cases, wherein we have determined from such physical sign that mitral regurgitation existed during life, no lesion whatever indicating inadequacy of the mitral valve to close its orifice has been discovered after death. Moreover, in some cases where we have not only observed the sign mentioned, but where the whole category of signs, symptoms, and consecutive changes which experience has taught us to associate with mitral inadequacy has been present, the autopsy has demonstrated no determinate lesion at the orifice.

It will best serve a practical purpose, I think, if we divide

the cases in which the signs indicating mitral regurgitation are evident, into clinical groups, discussing the bearing of the collateral phenomena upon treatment in each group. We shall thus consider the cases just as we meet with them in practice.

I. A case presents itself, manifesting signs indicating mitral regurgitation in the subject of marked *anæmia*. We have to inquire whether or no there has been antecedent disease leading up to organic change at the mitral orifice. Supposing such signs are not in evidence, have we a right to assume that actual mitral regurgitation can be induced by the condition of anæmia without concurring causes? The answer is, in my opinion, undoubtedly in the affirmative. In cases of anæmia and chlorosis a murmur is sometimes heard exactly in the site of that indicating mitral regurgitation. I have observed not only this sign, but all the concurring signs of cardiac failure, in a woman who suffered from excessive periodic hæmorrhages per vaginam, associated with uterine fibroids. I was called to such a case (Mrs. H., aged thirty-nine), manifesting severe dyspnœa such as one meets with in cardiac disease, extensive œdema, and a loud systolic murmur heard at the apex of the heart. The patient was very anæmic from copious hæmorrhage, the cause of such hæmorrhage having been diagnosed by Sir Spencer Wells to be uterine fibrosis. With care, rest, and suitable treatment, she recovered from all the symptoms denoting cardiac disease, and the murmur wholly disappeared. This I consider to have been an instance of what Professor Balfour has termed "curable mitral regurgitation."

In the disease known as *progressive pernicious anæmia* it is common to find an apex-systolic murmur. Such was noticed in four of eight cases recorded by Dr. Byrom Bramwell. In one an observer had diagnosed the case as cardiac dropsy

from mitral insufficiency.* In three cases recorded by my colleague, Dr. Stephen Mackenzie, an apex-systolic murmur was noted. Though in many of such cases the murmurs are heard at the base of the heart and over the site of the pulmonary artery, they are, as Dr. Stephen Mackenzie has said, "sometimes loudest at the apex of the heart, conducted into the axilla and heard at the angle of the left scapula. It is remarkable how loud and harsh these bruits sometimes are."†

A series of phenomena strictly analogous to those just mentioned as occurring in the human subject can be induced in animals by copious bleedings. Dr. Donald MacAlister says: "When an animal is bled till it is feeble, a murmur indicating regurgitation from the ventricle is heard with the heart-sounds. You may inject a proper saline solution to make up the normal quantity of circulating fluid, but still the regurgitation occurs. As the animal makes blood again, so that its muscles are again properly nourished, the murmur disappears."‡

And now, assuming that in these cases there is a veritable regurgitation, how is such brought about? The explanation is, I think, given by the careful experiments conducted by Ludwig and Hesse at Leipzig, which have been admirably summarised by Dr. Donald MacAlister.§ The mechanism for the closure of the left auriculo-ventricular orifice does not reside in the valve alone; the surrounding muscles of the ventricle have an active share not merely in floating up the valve-curtains, but in reducing the size of the aperture

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* *Edinburgh Medical Journal*, November, 1877.

† "Clinical Lecture on Idiopathic, Essential, or Pernicious Anæmia," *Lancet*, 1879.

‡ *British Medical Journal*, October 28, 1882, page 825.

§ "Remarks on the Form and Mechanism of the Heart," *loc. cit.*

which these valve-curtains have to close. In Dr. Mac-Alister's words: "As systole begins, the muscles surrounding the ostia contract; and presently, instead of the round gaping orifices of diastole, the valves have to close oval and compressed ones. . . . . The base muscles do their share of the work of closure, the valves promptly complete it." When the muscles of the base are enfeebled, as in the cases which we have been considering, the valve-curtains are insufficient to close the orifice because such orifice is wider than usual. It is not that the aperture is dilated, but that it is insufficiently contracted, the aid of the muscles which normally produce such contraction being lost.

Regurgitation may result, therefore, from feebleness of muscle, and restoration to the normal may occur with improved nutrition; but it must be recollected that persistent anæmia or repeated blood-lettings (as shown by experiments on animals) will induce a fatty degeneration of the heart-muscle, a morbid condition which may be irrecoverable.

I think it will be agreed that both for prognosis and treatment it is important that we should be able to make the differential diagnosis between a regurgitation due to feebleness of muscle, the result of anæmia, and organic disease at the mitral orifice. I will suppose that in a case of anæmia presenting a systolic murmur at the apex there is no evidence to lead us to suspect previous valvular disease, and no history of rheumatism. It may be, however, that the regurgitation is not from adynamia of the ventricle, but from an endocarditis of insidious origin, such as I have previously described. Can we rely for guidance on the physical signs? I will mention an illustrative case. I was called a short time ago to a patient at the London Hospital, who was supposed, after the preliminary examination, to be suffering from mitral

disease. There was a loud apex-systolic murmur, typical of mitral regurgitation. On delineating the outline of the heart by percussion, however, I noted that there was no notable dilatation such as one would expect to find in organic heart-disease when failure was imminent; for the patient was extremely ill. Noticing the very marked pallor, I suspected that this might be a case of idiopathic pernicious anæmia, and in confirmation of this view I found the fundus oculi studded with abundant hæmorrhages. I have no doubt, both from these reasons and from the clinical history, that this was a case of mitral regurgitation in association with pernicious anæmia. Unfortunately, the patient being a Hebrew, an autopsy was not performed. I would insist, therefore, on the value of *determining the outline of the heart by percussion* as a means of differential diagnosis in these cases. In anæmia, as I have observed, the heart is not notably dilated.*

In the case I have mentioned as occurring in conjunction with hæmorrhage, I found the *determination of the tension in the arterial pulse* to be a very important means of differential diagnosis. In advanced organic mitral disease—when, for example, as in the case cited, dropsy and extreme cardiac dyspnœa have supervened—the arterial tension is usually low (Fig. 8). In the case mentioned I found the opposite indication—the tension, as shown by the sphygmographic tracing, was rather high. It is an unexpected thing, as Dr. Broadbent has pointed out, that "in a disease such as chlorosis, characterised by debility, there should be high

* Dr. Allbutt has found that in progressive pernicious anæmia the heart is not dilated, but simply atrophic. Dr. Theodore Williams has observed that some cardiac hypertrophy often follows anæmia, but dilatation is not evidenced. (*Cf.* discussion on Professor Balfour's paper, "Arguments in Favour of Dilatation of the Heart as the Cause of Cardiac Hæmic Murmurs, etc," *British Medical Journal*, August 26, 1882, page 354.)

arterial tension: but such is the fact."* My experience is in this particular entirely in accord with Dr. Broadbent's.

These two signs, therefore—an area of cardiac dulness not perceptibly greater than the normal, and a heightened tension in the systemic arteries,—I consider to be of the greatest importance in differentiating in a very anæmic patient between organic disease at the mitral orifice and incomplete closure from adynamia.

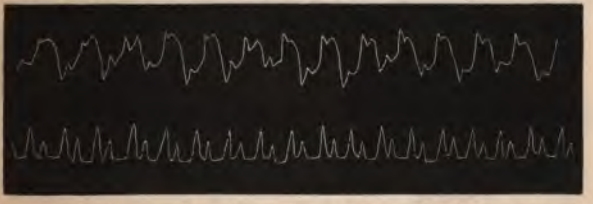

FIG. 8.—Cardigram and sphygmogram in a case of advanced mitral disease, with free regurgitation.

As regards treatment, such differentiation is important for I have never known, in the class of cases we are now considering, any marked improvement follow the administration of the usual cardiac tonics, such as digitalis and iron. In the cases attended with hæmorrhage, it is, of course, of the first importance to arrest this at its source. Rest, and the administration of assimilable food, are no less important indications. In this connexion I may call attention to the great value I have observed to attach to *supplementary alimentation by the rectum* in such cases. I have long tried the plan of using defibrinated ox-blood for a nutrient enema, as advocated by my friend, Dr. A. H. Smith, of New York. In comparing results, however, with those in which artificially digested food has been employed, I consider that the

* *British Medical Journal*, August 26, 1882, page 355.

balance of evidence is in favour of the latter plan. I have had prepared mixed peptone enemata—beef, milk, and farinaceous food—which have been proved to preserve in perfectly good condition for long periods. These have the advantage of being available at a moment's notice, it being only needful to render them diffluent with warm water. From two to four ounces are injected slowly into the rectum, and repeated every three or four hours. In many cases I have caused to be added the dried ox-blood (*sanguis bovinus exsiccatus*), in the proportion of a drachm to the ounce. I have lately, however, adopted a simpler plan with good results—using, instead of peptoned food, equal parts of warm milk and cod-liver oil as a nutritive enema.

In the treatment of cases of idiopathic anæmia, I have found no drug-treatment so efficient as the administration of arsenic (Fowler's solution in small doses, gradually increased). I have observed, as has been recorded by others, complete recovery with the disappearance of the cardiac murmur under such treatment, combined with rest and careful nutrition.

It has been supposed by Naunyn, Balfour, and others that actual regurgitation through the mitral orifice is in existence in cases where a systolic murmur is observed in the second left interspace near the border of the sternum. I am far from convinced that such view is correct, and prefer to adhere to the opinion that such murmurs are generated usually in the pulmonary artery. A consideration of this debateable question is unnecessary here, as I am dealing with those conditions in which observers would generally agree that mitral regurgitation was undoubtedly indicated.

II. We will now assume that a systolic apex-murmur is present in a patient showing signs of *a neurosis of the cervical sympathetic*. It has been frequently noted that

a murmur at the apex has existed in the subjects of exophthalmic goître (Graves' or Basedow's disease); yet, on post-mortem examination, no disease at the mitral orifice has been discovered. In these cases anæmia may be present, but not of necessity. It is not causally related with the phenomena. Organic heart-disease may co-exist, but such coincidence is rare. It is important to recognise—especially with regard to treatment—that in the subjects of Graves' disease mitral regurgitation occurs without valvular lesion. I now wish to draw attention to a point with reference to this curious affection, viz., that, as I have myself observed, the triad of symptoms—the protrusion of eyeballs; the thyroid enlargement; the paroxysmally disturbed, rapid, palpitating heart—can be disunited: and we may observe in a given case a union of two of the groups, or even one group alone. For example: I lately brought before the Ophthalmological Society a patient manifesting pronounced exophthalmos without thyroid or cardiac symptoms. I have lately seen in consultation a case manifesting only the cardiac phenomena, the heart's action being very rapid and the paroxysms of palpitation extreme. Again, I have lately observed the case of a lady in whom there is a combination of the cardiac and thyroid symptoms without exophthalmos. In each of these cases there was a history of shock, mental anxiety, or nervous exhaustion as a proximate cause. In the last case the cardiac trouble was severe besides distressing paroxysms of palpitation a loud murmur was manifest at the apex, and extensive œdema supervened. In fact, the case closely resembled one of organic mitral disease. There can be but little doubt, I think, that in these cases there is disorder, if not disease, of certain ganglia of the cervical sympathetic. The record of fatal cases in which such disease has been actually demonstrated is now

tolerably extensive. Trousseau, Cruise and McDonald, Reith and Shingleton Smith, have recorded cases in which some of the ganglia (usually the inferior cervical) have been enlarged, atrophied, or degenerated. Such observations have a distinct bearing on treatment. In the cases which I have seen, ordinary tonics and digitalis have been of very little benefit, but great improvement has followed galvanisation of the cervical sympathetic. I have employed the continuous current, from twenty to forty elements (Léclanché). One pole may be placed behind the lower jaw in front of the sterno-mastoid, and the other either at a corresponding point of the opposite side, or at the nape of the neck right or left of the vertebra prominens, or above the sternum at the inner edge of the insertion of the sterno-mastoid.*

III. I now turn to a third group of cases, and assume that the indications of mitral regurgitation are manifest *during the evolution of certain fevers.* In the course of typhoid fever, for example, a systolic murmur may be discovered at the apex. There is no history of its existence before the attack, but it has arisen during the course of the disease. M. Hayem has especially studied these phenomena. He says, "In the course, or at the end, of the second week there arises, in a certain number of patients, a bellows murmur with the systole. At the time of its first appearance this murmur may be soft and of little intensity. Its maximum is at the apex in the neighbourhood of the nipple, but it is prolonged towards the base, becoming feebler there. Often this bruit has an intensity and roughness equal to

* Such treatment has been recorded as successful by von Dusch, Chvostek, Moritz Meyer, Eulenburg, Guttmann, Remak, Ancona, and others. *Cf.* Althaus, "Medical Electricity," third edition, pages 185 *et seq.*, 335, 621; Hayden, "Diseases of Heart and Aorta," pages 1050 *et seq.*; Ancona, *Giornale Veneto delle Scienze Mediche* (*British Medical Journal*, June 1, 1878, page 790).

organic mumurs; or at first of only slight intensity, it may soon become louder, and make one believe in the existence of endocarditis. Moreover, it may vary in intensity from day to day, or may become modified by a change of position of the patient, as one may observe when auscultating in the lying and sitting positions alternately."* In typhoid fever, therefore, it may be an important question as to the nature of such a murmur, and its bearing on treatment. The clinical evidence shows that in the course of the fever the murmur changes its site and fades away, and that it may be accompanied by reduplication of heart-sounds and disturbances of cardiac rhythm. Thus, in the case of a young lady aged nineteen, observed by myself, there appeared, on the eleventh day of typhoid fever, a soft systolic murmur, left of the sternum, at the third costal cartilage; on the thirteenth day the bruit extended nearly as far as the apex; on the fifteenth it reached the apex; on the seventeenth it was right of the apex, and there was reduplication both of the first and second sounds; on the nineteenth, twentieth, and twenty-first days reduplication of the first sound only was heard, the murmur having disappeared.

The murmur, therefore, is an evanescent one. To what is it due? The changes are, according to M. Hayem's observations, not in the endocardium nor pericardium, but in the muscle of the heart. In fatal cases the muscular fibres present a granular and fatty degeneration, or a special form of *vitreous* degeneration; the areas of morbid change are disseminated in an irregular manner here and there throughout the cardiac muscle. There are, besides, a multiplication of the muscular nuclei and aggregation

* *Cf.* "Des Manifestations Cardiaques de la Fièvre Typhoide," par M. G. Hayem: *Le Progrès Médical*, 17 Juillet, 1875, page 401 *et seq.*

of cellular elements. In fact, the disease is a form of myocarditis.

It is, I think, sufficiently proven that the murmur occasionally heard at the apex in cases of typhoid fever is due to regurgitation on account of imperfect apposition of the valves of the left or right sides from enfeeblement, by disease, of the muscular fibres in certain areas of the heart-wall. It does not appear that the occurrence of such murmur renders the prognosis more grave; but sudden death, in all probability from myocarditis, may occur in typhoid without any special evidence of direct cardiac impairment previously. Its occurrence, however, should make us watchful, and cases presenting any of the phenomena indicating myocarditis in typhoid should be observed, and treated with a view of preventing subsequent dilatation.

Analogous myocarditis has been described in variola (by MM. Desnos and Huchard),* and in severe forms of intermittent fever as observed in Africa by M. Vallin.†

It is obvious that a recognition of the nature of the alteration which produces a mitral regurgitant murmur in the cases we have been considering must have an important bearing on treatment. We need not fear that endocarditis has arisen as a complication, nor have we to debate as to an anti-rheumatic plan of treatment. The indication is to keep the disturbed muscle of the heart as tranquil as possible, and of course to promote as good a nutrition as the circumstances will permit.

IV. I now come to the fourth group, and assume that a murmur indicating mitral regurgitation is observed in the subjects of *acute or subacute rheumatism*. Attention has

* "Des Complications Cardiaques dans la Variole, et notamment de la Myocardite Varioleuse," *Union Médicale*, 1870-71.

† *Union Médicale*, 1874, pages 293 and 316.

been frequently drawn to the fact that murmurs may arise in the course of evolution of the disease, and yet disappear, and patients being free from murmur have been considered to be free from cardiac complication. I have in my former lecture deprecated this as a too hasty conclusion. It may be well to inquire, in the first place, what is the probable nature of these transitory or evanescent murmurs, which are by no means uncommon, for they occur, as the statistics of the London Hospital for 1880 and 1881 show us, in about 10 per cent. of the cases. Rheumatism is a disease notably attended with anæmia. Is it probable that these bruits are of the nature of those which we have considered to be causally related with anæmia? The evidence collected for me by Dr. Gabbett as to the site of such transient murmurs is, I think, against this view. It is well known that the murmurs heard in connexion with anæmia, though sometimes heard at the apex and indicating mitral regurgitation, are far more frequently audible at the base over the site of the pulmonary artery or aorta. Even when heard at the apex they are usually accompanied by other murmurs at the base. In rheumatism, however, the usual site of the evanescent murmur is the apex. The totals for 1881 show as follow:—Transient murmurs in mitral area fifteen, at base and apex seven, in aortic area five, in pulmonic area three. It would appear that a murmur which might suggest an anæmic causation is almost confined to a first attack of rheumatism; after two or more attacks no basic transitory murmurs are recorded. Then as regards the transient systolic murmur in the mitral area, we may ask whether it may be due to myocarditis. If so, it does not resemble in associated phenomena the murmur observed in typhoid, etc. The peculiar perturbations of rhythm are not recorded, and it would appear probable that if there be myocarditis it does

not occur in disseminated areas as in typhoid. May it not be that the temporary regurgitation is due to a localised myocarditis, developed in the neighbourhood of the swollen valve or inflamed endocardium? Thus, though the swollen valve might not be in itself incompetent, a temporary incompetence would be produced by the impairment of the force of the muscle. As the myocarditis subsided the valve would become again competent, but probably, in many instances, to present a renewed imperfection when the swelling in the course of time had given rise to fibrous change and consequent retraction. I draw attention to this as a caution as to the expression of my opinion that a valve is sound after a murmur developed during rheumatism, though the murmur be temporary.

Let us now suppose that owing to rheumatic endocarditis the mitral valve has been rendered incompetent. It is well known that such may be the case, and yet the subject of such incompetence present no sign nor symptom of deviation from health. We are familiar with cases manifesting the murmur of mitral regurgitation in childhood, who pass through the period of adult life without suffering from the distresses of cardiac disease, and who, perhaps, ultimately succumb to an affection, the course of which the valvular imperfection has in nowise sensibly modified. In such cases the valvular imperfection has become *compensated*.

Supposing a regurgitation just instituted, the first effect is upon the left auricle, which is now made to contain a quantity of blood greater than normal by so much as gushes into the auricular cavity at each systole. The effect is to distend and to dilate the auricle. The left ventricle, too, is filled more rapidly than under normal conditions, because the blood from the auricle enters it under pressure the

moment that diastolic relaxation permits. Such entrance of blood is more free than the normal. Hence dilatation or hypertrophy of the left ventricle, or a *tendency* thereto. The most important of the induced conditions is, however, that of the pulmonic circulation. The reflux current overfills not only the auricle, but the pulmonary veins and the pulmonic capillaries. Against such resistance comes the force of the right ventricle in systole, which, in opposing the resistance, becomes hypertrophied. The hypertrophy of the right ventricle is essentially conservative, and the increased tension in the pulmonic circulation is an essential condition of compensation. The sign of such heightened tension, and therefore compensation, is, as long ago pointed out by Skoda, accentuation of the pulmonic second sound in the second left interspace.

Observation of the degree of pronunciation of the pulmonary second sound is of the highest importance as regards the treatment of mitral regurgitation. It is in a considerable degree a measure of the amount of such regurgitation. If the aperture caused by incomplete mitral closure in systole be small, the pulmonic tension is only slightly increased, and the pulmonic second sound may not be perceptibly intensified; but if the gap be wide, the tension, supposing the two ventricles to be in an efficient condition of compensatory hypertrophy, is great in the pulmonic circuit, and the second sound in the pulmonary area is very loud. If afterwards the loudness of such second sound is found to diminish, such sign is of very high importance. It suggests that the compensatory hypertrophy of the right ventricle is beginning to fail, that dilatation is in excess, and that the tension of the blood in the pulmonary artery is reduced by so much as regurgitates through the tricuspid orifice. Of course, the other signs of tricuspid regurgita-

tion should be taken in conjunction with this, but I know no sign which is so valuable a guide for treatment.

As the left auricle is overfilled in proportion to the amount of blood regurgitating, so is the aorta, and from it the systemic arteries, ill supplied. A diminution occurs in the normal quantity of blood propelled to the tissues, while in the veins circulation is retarded, and the normal content is augmented. There are arterial anæmia and venous plethora. The institution, however, of compensatory hypertrophy of the right ventricle rectifies the ill supply to the aorta. The increased pressure in the pulmonic circuit at the time of systole opposes the reflux into the auricle, and the current thus opposed is urged in normal amount into the aorta. So, even supposing that the force of the left ventricle be not augmented, increased force of the right may restore the equilibrium by inducing a pressure in the auricle equivalent to that afforded by a competent valve.

As regards the mode of production of compensatory hypertrophy, I would draw attention to an excellent chapter in Dr. Milner Fothergill's work.*

The practical question which it becomes us to answer, when a patient comes before us who presents signs of mitral regurgitation the legacy of rheumatic endocarditis, is—Is this valvular imperfection duly compensated or not? Subjective symptoms may tell us of such want of compensation, but they are often deceptive. In addition to the auscultatory sign I have mentioned, we may get valuable evidence from the use of the sphygmograph and cardiograph. The former may tell us of a fairly normal tension in the systemic arteries or otherwise; the latter, by recording the duration of systole and diastole, may inform us how far

* "The Heart and its Diseases, with their Treatment," second edition, chapter v., page 96. London: H. K. Lewis. 1879.

the normal rapidity of filling of the ventricle is exceeded, and thus may give evidence of the amount of regurgitation.

Supposing that we are satisfied that there is due compensation, medicinal treatment may be entirely unnecessary. I have no doubt that a vast amount of injury has been done to patients by a shaking of the head of the auscultator over the subject of a mitral murmur, who, perhaps, was no worse at the time of examination than he was ten, twenty, or thirty years before, and who might continue uninfluenced for harm by his cardiac complication all his days. He should be cautioned against strain, against exposure, and against irregularities of diet, etc.; he may be better occasionally for treatment by iron tonics, cod-liver oil, or strychnine, but any special *cardiac* treatment is out of place.

Not so, however, if there is evidence that compensation is beginning to fail. I will pass in brief review the chief agents which are of service in such case.

(1.) Digitalis is *facile princeps* of drugs in the treatment of imperfect compensation. The researches of modern observers—Fuller, Handfield Jones, Ringer, Balthazar Foster, Traube, Wood, Lauder Brunton, Milner Fothergill, and others—have shown its mode of action; that it so influences the cardiac ganglia as to induce a more perfect contraction of the ventricular muscle, and hence a more complete emptying of the ventricles; whilst, at the same time, by an action on the vaso-motor centre, it causes contraction of the arterioles and a heightened tension in the arterial system. It slows the heart by lengthening the diastolic pause; so not only does it give rest to the wearied cardiac muscle, but—as this muscle is nourished only during such diastolic pause by the blood which then enters through the coronary arteries—it directly ministers to its nutrition. It is a matter of common experience how that digitalis,

especially when combined with iron, strychnine, cod-liver oil, and other tonics, restores the *status quo ut ante* when, in a patient manifesting a mitral systolic murmur, the evidence shows that compensation is beginning to fail. As, however, with every other medicinal agent, caution must be used in the administration. As regards *dosage* a certain golden mean has to be observed. The often repeated maxim concerning the middle way points its lesson again :—

"Levis alit flammas : grandior aura nocet."

A little over a suitable dose may induce nausea, vomiting, anuria, irregularity of pulse, and, instead of slowing, an enhanced rapidity of heart's action. Whilst a dose which produces a favourable result is constant and discoverable in regard to a large majority of patients, in a minority such dose is inconstant and even unattainable.

As regards the preparation used, we may have differences of result; and we know that, as in the case of so many vegetable products, the energy of different samples may vary. The pharmacopœial equivalents of the officinal drugs P.B. are as follow :—

One grain of the dried and powdered leaves = one-third of an ounce of the infusion = eight minims of the tincture.

Practically, I consider the tincture most reliable, and that usually in small doses (ℳv. to ℳx., increased only in exceptional cases, and then occasionally reduced). Next in value I esteem the powdered leaves (gr. ½ ad gr. ij.), the combination of which with alkalies is very useful.

In some cases, even by increasing the dose no apparent influence appears to be exerted by the drug : then digitaline, especially when hypodermically injected, I have observed to give in many cases good results. The digitaline hitherto prepared has probably scarcely ever been the pure

alkaloid; but it appears likely that by a new process it can be obtained in a state of purity. The usual dose for hypodermic administration is one-fiftieth of a grain. In a child of ten years of age, with dropsy and great distress from mitral regurgitation, I found, after each injection of one-hundredth of a grain of digitaline, hypodermically, at intervals of four hours, the pulse-rate reduced by eight per minute almost immediately. In this case recovery took place from the urgent symptoms, and the child was sent to a convalescent home. She relapsed, however, and died three months afterwards when away from our observation.

When the right ventricle has dilated so far that there is marked tricuspid regurgitation, the beneficial action of digitalis is by no means so decided. Nevertheless, in some cases, especially when occasional purgation is a part of the plan of treatment, the signs of tricuspid regurgitation may pass away. For instance, in a child (Alice B.), aged eleven, under my care at the North-Eastern Hospital for Children, mitral regurgitation with dropsy was manifested, and marked venous pulse was seen in the left external jugular. Treatment consisted of six-minim doses of tincture of digitalis three times a day. The child had taken previously, as an out-patient, four-minim doses with four grains of ammonio-citrate of iron three times a day. After twenty-one days all severe symptoms had passed away; there was no venous pulse, and the case was discharged as convalescent two days afterwards.

In other cases no such favourable result attends. In fact, as *à priori* consideration might suggest, any increased power of systole which the digitalis may bring about serves the more to force back the blood through the imperfect tricuspid orifice into the venous channels. But yet I have seen good results when the administration of digitalis has been com-

bined with abstraction of blood by leeches or cupping. In a child of ten (Maria W.), manifesting mitral and tricuspid murmurs with percussion-evidence of greatly dilated right ventricle, after rest in hospital for a fortnight and administration of tincture of digitalis in four-minim doses with tincture of the perchloride of iron (ℳx.), and a single leech applied to the epigastrium every other day for fourteen days, it was noted that the dulness over the right cavities receded to the mid-sternal line coincidently with general signs of amendment. I prefer very small abstractions of blood, repeated every two or three days, to larger bleedings at longer intervals. In a case lately under my care at the London Hospital this lesson seemed to be pointed, though the recovery was very satisfactory.

Alice F., aged eleven, was under my care for mitral and tricuspid regurgitation, with great and advancing œdema, orthopnœa, and cardiac distress. She was treated by twenty-minim doses of tincture of perchloride of iron, with five minims of tincture of digitalis. After twenty days, tincture of casca was substituted for digitalis, with no apparent benefit. Digitalis was then resumed as before, and, considering the great distension of the right chambers, six leeches were applied to the chest. Great relief of dyspnœa followed, and œdema became less. Improvement was maintained for ten days, and then urgent dyspnœa and signs of greater dilatation of right chambers occurred. Casca was again tried, and this time with some apparent benefit. Purgatives (pulv. jalapæ co. ℥ss.) were also administered, but still the grave signs of right-ventricle engorgement continued. Again six leeches were applied to præcordium. A few days afterwards the right subclavian vein was found to be plugged, and the whole arm and forearm became enormously swollen. It seemed to me possible that the abstraction of

blood, by rendering coagulation more easy, had perhaps disposed to the thrombosis. Nevertheless, I was convinced that the cardiac trouble was sensibly relieved by the leeching, and this was repeated, and saline diuretics and digitalis again administered, in doses increasing from ℥v. (℥vij., ℥ix., ℥x., to ℥xx.). Under this treatment there was gradual but very marked improvement. After five days of the full dose of digitalis it was altogether omitted for ten days, and then resumed in ten-minim doses. All the urgent symptoms passed away, the enormous swelling of the arm due to the venous thrombosis entirely subsided, and the patient was discharged convalescent and able to walk with comfort after having been in hospital for six months.

(2.) *Belladonna* is, I think, only useful in the treatment of failure of compensation in cases of mitral regurgitation when combined with, or *occasionally* substituted for, digitalis. Belladonna, like digitalis, increases the power of systole and raises the arterial tension. As Dr. Lauder Brunton has shown, it paralyses the cardiac terminals of the vagus, and reduces irritability by an anæsthetic effect on the sensory nerves of the heart. Very useful occasionally, it by no means compares with digitalis for prolonged employment. The hypodermic injection of one-fiftieth of a grain of digitaline, with one-sixtieth of a grain of atropine, I have found very satisfactory.

(3.) *Casca.*—A tincture made from the bark of *Erythrophlæum Guinense*, the ordeal bark of West Africa, has been employed as a substitute for digitalis. Dr. Lauder Brunton, in his Gulstonian Lectures for 1877, published the results of elaborate experiments as to its physiological action. In kind this action appears much to resemble that of digitalis. Dr. Brunton has said, "Digitalis has hitherto been our great resort in mitral disease, but I think it probable that

in casca we possess a drug more powerful still; at least, its effect upon the arterioles appears to be greater than that of digitalis, and it is quite possible that it may succeed in those cases of advanced mitral disease where digitalis fails." I have myself employed the tincture of casca, substitutively for digitalis, in a considerable number of cases, but I have never yet been able to convince myself that it has any more beneficial action in mitral disease.

(4.) *Caffeine.*—Gubler, Shapter, Leech, Milliken, Brakenridge, Huchard, and others, have recorded observations showing the action of caffeine (or its citrate) in cases of cardiac disease, especially where dropsy is a marked symptom. Some of the cases show very forcibly that a beneficial influence has been exerted by the drug. There are many apparently contradictory *data* as to its physiological action, but the cardinal points are, that it first quickens, but soon after slows the heart's action, that it increases the general arterial tension, and acts in a very pronounced manner as a diuretic in cardiac dropsy. Dr. Brakenridge advises that digitalis be administered previously to, or in conjunction with, the citrate of caffeine, and that small doses (three grains) should be employed.* M. Huchard, however, recommends that caffeine, and not its citrate, should be used, and that in larger doses—four to six grains.† It produces diuresis more rapidly than digitalis, and has none of its nauseating effect. I have employed citrate of caffeine in substitution for digitalis without any marked benefit being manifest; indeed, I have found that in some cases it has induced insomnia. Nevertheless, I consider that the evidence is such that I shall certainly employ it in any case where, in cardiac dropsy, a rapid diuretic effect is desirable.

* *Edinburgh Medical Journal*, July and August, 1881.

† *Bulletin Général de Thérapeutique*, Paris, 30 Août, 1882, page 145.

(5.) *Convallaria Maïalis.*—This is the well-known lily of the valley, long employed by the Russian peasantry as a remedy for dropsy. It is botanically closely allied to asparagus, the diuretic effect of which is well known. M. Germain Sée has made a series of researches which point to it as probably a valuable agent in the treatment of failure of compensation in cardiac diseases.* The preparation used is an extract of the whole plant—flowers, stems, and roots. The mode of action of the extract of convallaria also resembles that of digitalis: it slows the heart whilst increasing the force of systole, and augments arterial tension. It is said that it does not, like digitalis, exhaust the contractibility of the heart and arteries. Administered in doses of fifteen to twenty-two and a half grains during the day it has apparently produced very favourable results. M. Sée has recorded five cases of mitral regurgitation in which it was employed. It entirely relieved the œdema and cardiac distress, and manifested a pronounced diuretic action. I am now employing the extract of convallaria, in mitral disease, in five-grain doses. I am convinced of its power of raising the intravascular pressure, and of its increasing the force of systole, but I am not yet convinced of its superiority to digitalis. The results, however, are such as to warrant an extended trial.

(6.) *Morphia.*—Judiciously employed, I consider that this is one of the most valuable of agents, or rather adjuncts, in the treatment of the distress, especially the dyspnœa and insomnia, attendant upon failure of compensation in cases of mitral regurgitation. I am strongly of opinion that it should not be administered by the mouth, but by hypo-

* "Sur un Nouveau Médicament Cardiaque: Recherches Expérimentales sur le Muguet (Convallaria Maïa'is)," par le Professeur Germain Sée: *Bulletin Général de Thérapeutique*, Paris, 30 Juillet, 1882.

dermic injection. When given by the mouth it disagrees, just as opium frequently does; whereas, administered hypodermically, it calms the most distressing dyspnœa, without inducing, so far as my experience goes, any ill effect. The value of the hypodermic use of morphia in the distress of heart disease was brought before the profession, in his usual forcible and able way, by Dr. Clifford Allbutt in 1869.* I entirely endorse his view of the value of the remedy and its innocuousness in cardiac failure. I have found it valuable to combine the morphia (usually a hypodermic dose of one-third of a grain) with atropine (one-sixtieth of a grain), or digitaline (one-fiftieth of a grain), but the morphia is an essential agent for the successful treatment of mitral regurgitation when there is much respiratory distress coincidently.

In addition to such special cardiac treatment, general measures should be adopted for securing improved nutrition. The heart-muscle must not only be preserved from wasting, but it must also be fed. The problem of administering a due amount of nourishment is often a difficult one. Dr. F. T. Roberts has recommended in the gastric crises of cardiac disease, when there is an almost complete inability to take food, the use of peptonised aliment in a sipping fashion.†

This plan I would combine with the administration of nutritive enemata, as I have before mentioned. I feel sure, from my experience, that lives may be prolonged and crises tided over by such supplementary alimentation.

The foregoing is a brief sketch of the most important agents now at our disposal for restoring the power of the

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* *Practitioner*, 1869, vol iii., page 342.

† "Lumleian Lectures," *cf. British Medical Journal*, May 8, 1880, page 684.

heart-muscle and inducing the compensation in mitral regurgitation occasioned by rheumatic endocarditis, when failure threatens. The restoration of such compensation may not, however, be the only indication. Accidents of the disease, so closely related therewith as to force the necessity of considering them in any question of prognosis and treatment, demand consideration. Such epiphenomena are renewed attacks of endocarditis, pericarditis (especially when accompanied by myocarditis and adhesions), and embolism. These subjects, however, being equally manifest in mitral stenosis and mitral regurgitation, may be conveniently postponed till the next lecture.

V. There is, however, yet another group of cases of mitral regurgitation to consider. In these there is no history of antecedent rheumatism, the modes of causation we have hitherto discussed are excluded, the condition has arisen gradually in *association with conditions of high tension in the arterial system.*

The differentiation of this class of cases is important both for prognosis and treatment. A considerable minority of cases manifesting mitral regurgitation come to us with no history whatever of rheumatism. I calculate from the hospital records that these are about one-fourth of all the cases. I have said, however, in my former lecture that rheumatic endocarditis may be established without other evidence of rheumatism. It is obvious, therefore, that such cases of insidious endocarditis inducing mitral insufficiency may be included in the minority we are considering. The diagnosis between these and the cases of regurgitation due to yielding of the ventricle from intra-arterial pressure may generally be arrived at without difficulty. In the latter the signs are those of hypertrophy rather than dilatation of the left ventricle. The aortic second sound, if there be no evi-

dence of aortic disease, is pronounced, whilst (the amount of blood regurgitating being small) the pulmonary second sound is not accentuated. The arterial pulse is strong and incompressible, and often the arteries are tortuous and visibly atheromatous. It is obvious that these signs are very different from those usually met with in the rheumatic cases. Very important evidence is afforded by the sphygmograph, the pulse-trace showing in the non-rheumatic cases an ample tidal wave and the usual evidences of high arterial tension. Such cases are often associated with gout or with chronic renal disease.

In their treatment I have known no plan so successful as a protracted course of alkalies, with abstinence from alcohol, and as much rest as can be procured. Digitalis is not contra-indicated, notwithstanding the general high pressure in the arteries. I have found it of much service, probably by co-ordinating heart and arteries. In any of the accident of high tension, such as symptoms of angina or dyspnœa, occurring in this class of cases, the administration of nitro-glycerine or the inhalation of nitrite of amyl is of much service.

Lecture III.—MITRAL STENOSIS.

Morbid Anatomy—Physical Signs—Differentiation from the Lesion which induces Regurgitation—Rise and Progress of the Disease resulting in Stenosis — Compensation — Special Treatment in Mitral Stenosis—Complications of Mitral Disease —Pericardial Adhesions—Embolism.

I propose now to consider the morbid conditions associated with a structural change at the left auriculo-ventricular aperture—a change which narrows this outlet and impedes the influx of blood into the left ventricle during the period of diastole. No disorder of function can bring about such a condition as this; the lesions are always organic.

We will first glance at the *morbid anatomy* of the affection. If the mitral aperture be viewed from the auricle it may, in many cases, be seen that a smooth septum presents itself between auricle and ventricle, crossed by a narrow slit, almost straight, but inclining to be crescentic. Such slit may be no larger than a sixpenny-piece or a shirt-button will pass through, and from its appearance the orifice has been termed the "button-hole mitral." The natural form of the curtains may be entirely lost, their place being occupied by a thick fibrous structure welded at its circumferential attachment with the cords and fleshy columns, which may all be transformed into a dense tendinous mass. In certain cases this fibrous material is infiltrated with cal-

careous salts to such a degree as to make it closely resemble bone.

Another, but less frequent, form of obstruction is that in which the mitral orifice, as seen from the auricle, resembles a hollow cone. This is known as the "funnel-mitral." Its ventricular outlet may be so small that it will scarcely admit the point of the little finger. Dr. Hilton Fagge has recorded forty-six examples of the button-hole to one of the funnel form of constriction; Dr. Hayden, thirteen of the former to one of the latter; and of my own records of twenty autopsies in cases of mitral stenosis, two only were "funnel-mitral." M. Lancereaux has described a case of mitral stenosis in which, amongst the vegetations which surrounded the thickened orifice, he discovered hard granules, that were shown by chemical tests to consist of urates. When heated with nitric acid they gave rise to a yellowish product (alloxan), and this, on the addition of ammonia and distilled water, gave the characteristic red colour of murexide or purpurate of ammonia. The granules, when dissolved in acetic acid, crystallised in the characteristic rhomboids of uric acid.* I draw attention to this observation because it may have an important bearing on the questions of etiology and treatment. I have myself met with a case of mitral stenosis in which there were abundant gouty deposits in the joints, some of which suppurated, and gave exit to uratic concretions intermixed with the pus.

It is obvious that the great difficulty created by such alterations as these is the due filling of the ventricle from the auricle. In addition, there is, however, in many cases necessarily a reflux into the auricle at the systole of the ventricle. In proportion as the slit is narrow the possi-

* "Anatomie Pathologique," page 215. Paris: Victor Masson et Fils. 1871.

bility of such reflux is less, and in extreme cases of stenosis it appears probable that no regurgitation is possible. In all cases the main difficulty is the obstruction; that of regurgitation is subsidiary, though frequently co-existent.

It requires only a slight consideration to be convinced that quite a different set of conditions obtains in mitral stenosis from that manifest in mitral regurgitation. Morbid anatomy teaches us that in stenosis the left ventricle is usually not dilated; it has its normal capacity, or is even smaller than natural. We should expect so, for the difficulty is not that the ventricle is habitually overfilled as in regurgitation, but that it is insufficiently supplied owing to the imposed obstruction. When the left ventricle is observed to be dilated in the autopsy of a subject of mitral stenosis, it is probable that mitral regurgitation or disease of the aortic valves conduced to such a change. Upon the left auricle the consequences of mitral stenosis are very manifest. It is usually not only dilated, but hypertrophied. The wall of the auricle may be increased in thickness from its normal of about three-twentieths of an inch (Bouillaud) to a quarter of an inch or more. I have found it a quarter of an inch thick in a case of mitral stenosis in a child. On the other hand, it is occasionally found dilated rather than hypertrophied. In one case I found it extremely dilated, and the walls almost as thin as an ordinary visiting-card. The dilatation and hypertrophy of the left auricle are also in accord with *à priori* considerations, for the cavity becomes overfilled on account of the obstruction to its outflow, and the muscle has a heavier task than the normal in aiding the filling of the ventricle. When dilatation is in excess, it is through an unusual failure of muscular power.

I turn now from the morbid anatomy to the *clinical history* of mitral stenosis, and I shall have to crave your indulgence

if I seem to dwell too long upon points which may not appear at first sight to have a very distinct bearing upon treatment. I feel sure that I shall have your concurrence when I say that no disease is well treated that is misunderstood. We have a great deal to learn as to mitral stenosis; it is, I feel quite sure, in many instances, unrecognised—not from any fault in observers, but from their misfortune. It is only comparatively recently that our pupils could be taught in our hospitals the methods of discriminating between cases of mitral stenosis and those of mitral regurgitation, and it is unwise to conceal the fact that difficulties in such differential diagnosis do occur. But it has, to my mind, been too hastily assumed that the consecutive changes and collateral phenomena in the two conditions are so closely similar that a plan of treatment for the one is equally applicable to the other. I need not ask you to concur with me in deprecating the plea of "*Cui bono?*" It is our bounden duty to learn all we can of the disorder we have to treat, even if the immediate influence of such knowledge upon treatment be not so very apparent.

Let us consider the signs by which we may recognise the condition of mitral stenosis. 1. *The murmur.* This is heard in the neighbourhood of the apex of the heart, in the mitral area, but, according to my experience, usually rather to the right of the apex. It occupies the diastolic period—the long pause—usually the concluding portion of it, and then it terminates abruptly with the first sound. It is chiefly Dr. Fauvel, of Paris, and Professor Gairdner, of Glasgow, we have to thank for accurately describing this murmur and making it available for the practical purposes of diagnosis. The distinction between the murmur indicating mitral stenosis and that indicating mitral regurgitation is to be made partly by the character of the sound and partly by the rhythm. The

stenosis-murmur is usually of a rattling and rolling character, but its chief characteristic is its abrupt termination—it ends with a sudden stop, as the murmur of regurgitation *never* does. Even when the murmurs of stenosis and regurgitation are combined there is usually a spot in the neighbourhood of the apex at which the former is heard to stop suddenly, and the systolic murmur to "tail off" from it. The rhythm is determinable by ascertaining the relation to the second sound and to the impulse of the heart. In approaching the apex from the base one may be convinced of the commencement of the murmur after the second sound. Near the apex one may hear that the termination of the murmur is with the impulse of the heart as felt upon the chest-wall, or, where this cannot be determined, with the pulsation of the carotid in the neck. Such are, very briefly, the chief characters of the murmur which is so commonly known as the *presystolic* murmur that has been considered to be almost, if not absolutely, pathognomonic of mitral stenosis. And now as to its mode of production—a question which is really of practical importance. Some observers have considered it due to the muscular contraction of the auricle urging the blood through the stenosed aperture into the ventricle. It is well known that Professor Gairdner proposed the term "auricular-systolic" to denote the murmur, but he did not ascribe its production wholly to the muscular contraction of the auricle. Dr. Wilks * considered that the murmur might anticipate the auricular systole, that it might occur "not only during the contraction of the auricle, but also during the heart's diastole and pause." Dr. Galabin came to a like conclusion from the evidence afforded by the cardiograph. I am able to afford the crucial

* *Guy's Hospital Reports*, third series, vol. xvi., March, 1871.

proof of the view that the causation of the presystolic murmur may be independent of the auricle: *first*, because in many cases I have observed that, though there has been present a prolonged presystolic murmur commencing in the long pause almost immediately after the second sound, cardiographic evidence has shown the auricular systole to occupy its normal position just anterior to the commencing contraction of the ventricles (Fig. 9);* *secondly*, because I observed a case in which a murmur occupied at one time a portion of, and at another almost the whole of, the long pause; and the autopsy showed that the auricular systole could have

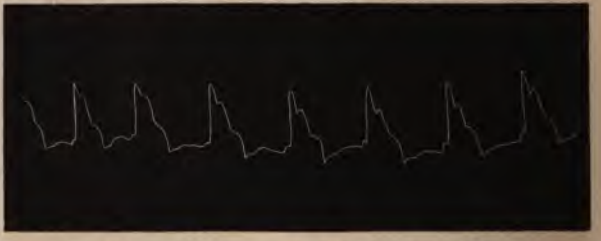

FIG. 9.—Cardiogram from a case of mitral stenosis with presystolic thrill and murmur, showing the auricular systole in the normal position.

had no share in producing such murmur—for not only was the left auricle so dilated that its wall could have exerted no appreciable muscular power, but it was lined by a closely adherent old laminated blood-clot. I consider that it is clearly proven that the so-called presystolic murmur may occur during the diastolic as well as the presystolic period, and that it may be due to the entrance of blood into the ventricle directly diastolic relaxation permits, the blood being urged through the stenosed aperture owing to the tension under which it has

* "Manual of the Physical Diagnosis of Diseases of the Heart," third edition, page 278. London: J. and A. Churchill. 1881.

been retained in the elastic and distended auricle and the pulmonary veins. The contraction of the auricle may reinforce the murmur and make it loudest just before the ventricular contraction. This consideration explains why, in exceptional cases, the murmur of mitral stenosis is post-diastolic and ceases with a distinct pause before the first sound, the auricular systole in such cases being weak or imperfect (Fig. 10). It is certain that in a large majority of

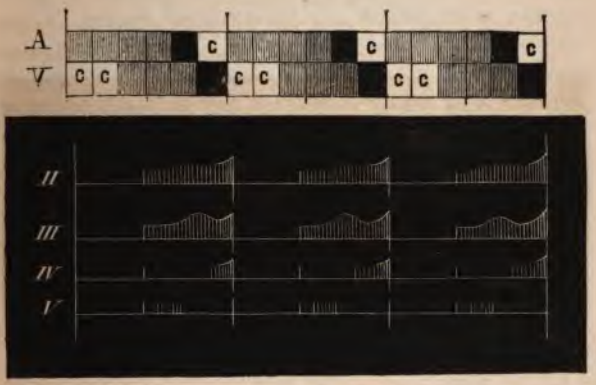

FIG. 10.—Diagram illustrating various forms of the murmur indicating mitral stenosis. II. Prolonged murmur from first to second sound. III. The same with reinforcement during its progress. IV. Presystolic murmur with reinforcement at its close. V. Post-diastolic murmur. A and V indicate the coincident phenomena in auricle and ventricle, the dark squares the periods of filling. C C, the systole or contraction.

instances the presystolic murmur serves to indicate with precision the existence of mitral stenosis. The late Dr. Hayden has said: "It is *never* present where mitral narrowing does not exist, and it is never absent, and that only for a very limited period, in cases of that lesion."*

* "Diseases of Heart and Aorta," page 898.

I am sorry that I cannot concur in so positive a statement. In a few cases I have found the presystolic murmur closely simulated by the murmur of aortic regurgitation, when this is conducted towards the apex, and especially, as is sometimes the case, when it is heard *only* in the mitral area. Cases have been recorded in which a presystolic murmur has been noted during life, and the autopsy has demonstrated not mitral stenosis, but aortic regurgitation. Another possible source of error is the existence of pericarditis, when friction may be occasioned by the auricle, and cease at the moment of systole. Again, I think, most observers will agree that in some cases the presystolic murmur is extremely variable: it may be inaudible during repose, and yet very evident when the patient is made to manifest some slight exertion; it may be absent for considerable periods, and then be readily discoverable. Although, therefore, I consider that in the great majority of cases the presystolic murmur declares with precision the existence of mitral stenosis, it is necessary to consider other signs before committing oneself to a positive opinion.

Another auscultatory sign of great importance in indicating the obstructive lesion is (2) *reduplication, or a seeming reduplication, of the second sound of the heart.* This phenomenon is to be noted in at least a third of the cases of mitral stenosis, and only rarely in other conditions. It becomes, therefore, a valuable aid to diagnosis. I have formerly developed before the Society at length my views as to the mode of production of this seeming reduplication.* I will only say here that I believe it to be due not to any want of synchronism in the closure of the aortic and the pulmonary semilunar valves, but to the normal second sound followed by another sound due to a sudden tension

* *Proceedings of the Medical Society of London*, vol v., page 191.

of the mitral valve itself. The blood, accumulated pressure in the auricle, rushes through the stenosed ture as soon as diastolic relaxation permits, and jerks the mitral curtains or the thickened material which represents them on the ventricular aspect (Fig. 11); this gives rise to a

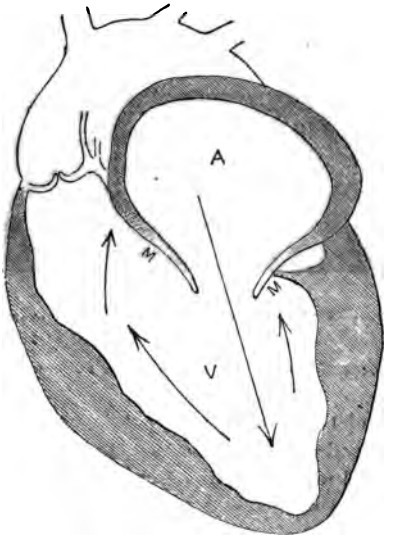

FIG. 11.—Diagram illustrating mechanism of pseudo-reduplication of heart sounds. Ideal section of left auricle and ventricle. M.M. curtains of mitral valve; the arrows show the direction of the flow of blood in diastole and auricular systole.

sound of tension, which, coming closely after the normal second sound, appears like a reduplication of the latter (Fig. 12). The great anterior flap of the mitral valve is normally on the stretch in diastole; in Dr. MacAlister's words, "it does not hang loosely down, it is stretched taut from basal ring

to muscle tip."* It does not seem difficult to realise that in the condition of stenosis, and for the reasons given, this diastolic tension may be so increased as to give rise to sound (Figs. 13, 14).

A third sign of importance in establishing the diagnosis of mitral constriction is (3) *thrill*. A thrill at the apex is rarely met with in mitral regurgitation, but very commonly in mitral stenosis. Its rhythm is determinable in like manner

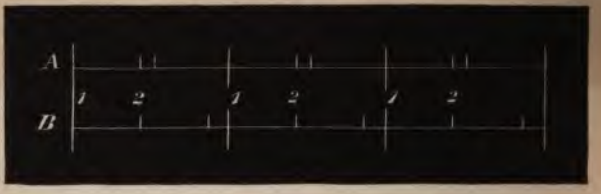

FIG. 12.—Diagram illustrating positions in cardiac rhythm of pseudo-reduplications of heart sounds. A, presystolic tension of mitral curtains occurring early, pseudo-reduplication of *second* sound. B, tension occurring late, pseudo-reduplication of *first* sound.

with that of the murmur, and if it be presystolic the diagnosis of mitral constriction is assured. I have observed presystolic thrill when there has been no presystolic murmur, and where the condition of stenosis has been indicated by other signs.

A fourth means of differentiation is (4) *the determination by percussion of the outline of the heart*. If this be done accurately by means of a pleximeter, and marked upon the chest-wall with a copying pencil, a transfer may readily be taken upon paper and kept for reference. By this method I have shown in some cases—(1) an abnormal bulging in the situa-

* *British Medical Journal*, October 28, 1882, page 825.

tion of the left auricle; (2) a dilatation of the right cavities and of the pulmonary artery, with an absence of dilatation of the left ventricle. The concurrence of these signs has strongly suggested the diagnosis of mitral stenosis when other signs have been obscure.

FIG. 13.—Cardiogram in mitral stenosis with pseudo-reduplication of second sound. The eminence *a*, indicating the systole of the auricle, is greatly exaggerated.

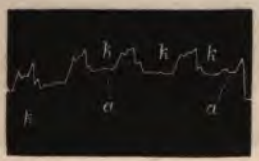

FIG. 14.—Cardiogram in pseudo-reduplication of second sound. Eminence *k* due to diastolic closure of semilunar valves is followed by an eminence *a*, indicating rise of blood-pressure in the diastolic portion.

Lastly (5), a valuable aid to diagnosis may be received from the *employment of the sphygmograph and cardiograph.*

Very contradictory opinions have been put forth as to the pulse of mitral stenosis. Dr. Hayden considered that "the pulse of mitral obstruction is usually quite regular, not above ninety in the minute, but small,"—that is, until the later stages, when failure commenced; and Dr. Fagge thought that in the majority of cases in which a presystolic murmur

was heard the pulse gave no indication of the existence of disease. A large number of observers, however, have noted irregularity of the pulse as pertaining to mitral constriction.* My own observations point strongly to a notable irregularity of the pulse in mitral stenosis; and this in such degree as to afford valuable diagnostic evidence. In mitral regurgitation the pulse is usually regular until compen-

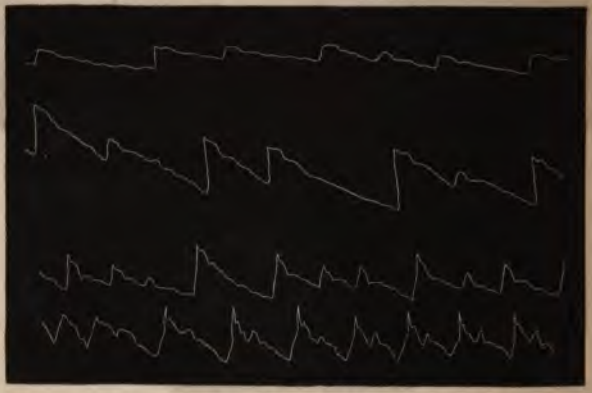

FIG. 15.—Sphygmograms in mitral stenosis showing interpolated systoles.

sation is beginning to be imperfect and the right chambers commence to yield. In mitral stenosis, however, irregularity may be evident when compensation is perfect. It is true that many observations may be made with a record only of an even and regular pulse; but with repeated observations the peculiarity of mitral stenosis becomes manifest in the trace—a double or even triple pulse is recorded before the base line of the sphygmographic trace is reached (Fig. 15).

* See list in Balfour's "Clinical Lectures on Diseases of the Heart." Second edition, page 123. London: J. and A. Churchill. 1882.

These pulsations are due to repeated systoles, the normal correlation between auricle and ventricle being disturbed. In the later stages, when the right side of the heart commences to fail, irregularities in volume of the pulse may be observed; and in a case where there was great dilatation of the auricle, I found the pulse become extremely slow, its rate falling from eighty to fifty-six, and then to an average of forty per minute. At one time it was thirty-six.

The evidence afforded by the cardiograph, when mitral stenosis is suspected, is, in my opinion, extremely valuable. The trace enables one to judge of the relative length of systole and diastole. In free mitral regurgitation a very short interval separates the systoles; the duration of the systole, instead of being, as in the normal, less than that of the diastole, is greater. In stenosis, on the other hand, the interval between the systoles may be greatly prolonged; or in stenosis the diastolic intervals may be observed to vary greatly in duration. Two systoles may occur with no appreciable diastolic interval, and another interval may be abnormally protracted. Much more characteristic, however, is the appearance of a number of vibrations in the diastolic part of the trace; in fact, the vibrations which are heard by the ear as murmur, or felt by the finger as thrill, may be written on the smoked paper by the needle of the cardiograph. I show you many examples. In some it will be seen that the diastolic portion is serrated, and there is no indication of the elevation caused by the auricular systole just before the main upstroke indicating the grasp of the ventricle; in others vibrations are seen to precede a defined systole of the auricle; in a third set the auricular systole is well marked, and the sonorous vibrations of murmur, though murmur existed, are not recorded. So I think we have a means of determining in some measure the degree of con-

striction. If such were considerable it is unlikely that the auricular systole would be readily transmitted and recorded; on the other hand, it is likely that the finely serrated line of vibrations would be produced by the extrusion of blood through the narrowed aperture.* Some of my tracings show in a marked manner the effect of effort in rendering evident vibrations in the diastolic portion which were not visible

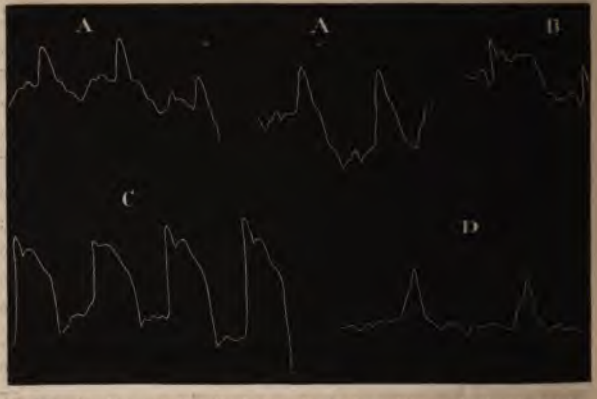

FIG. 16.—Cardiograms in mitral stenosis. A A, eminences indicating high blood pressure in diastolic period. B, pronounced auricular systole. C, D, irregular undulations and serrations in diastolic period.

during repose. By a comparison, too, of the characters of the systolic and diastolic portions I think we are enabled to obtain some indications whether, in combined stenosis and regurgitation, the former predominates over the latter or otherwise, and whether or no hypertrophy preponderates over dilatation of the ventricle (Figs. 16, 17, 18).

Such are the chief means at our command for arriving at

* In some instances there is cardiographic evidence of two or even three auricular systoles in one diastolic period.

a diagnosis of mitral constriction; and, though I do not think we are justified in coming to a conclusion from observation of the sign alone, I consider that, by a judicious

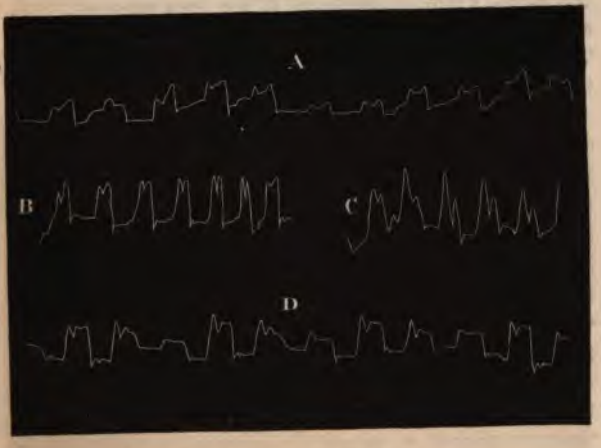

FIG. 17.—Cardiograms in mitral stenosis.

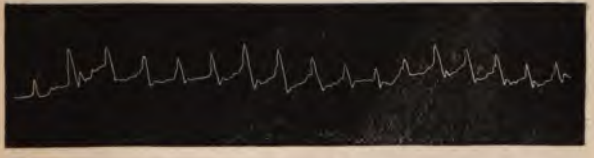

FIG. 18.—Cardiogram in mitral stenosis showing vibrations in diastolic period corresponding to presystolic murmur and thrill.

combination of methods of observation, no case of mitral stenosis ought to go unrecognised.

I pass on now to consider the clinical evidence as to the *origin and course of the morbid changes which bring about*

the obstructive lesion. We are at once met by a body of evidence which shows that mitral stenosis, like mitral regurgitation, has a strong relationship with rheumatism. From the morbid anatomy standpoint it has been supposed that, at least in some cases, the lesion might have been congenital. The smooth surface of the septum between auricle and ventricle, with its symmetrically edged aperture, might *primâ facie* support this view; but we do not find the lesion commonly associated with those which are undoubtedly congenital; and these are, moreover, infrequent in the left, though comparatively frequent in the right chambers of the heart. In one of the twenty post-mortems, however, which I have recorded, a large permanent foramen ovale was present, the subject being a female aged fifty. It is known that such congenital disease, as Dr. Peacock formerly pointed out, predisposes to endocarditis, and it is probable that such was the sequence in this case; for we are met by many observations which show that the lesions of stenosis, which in appearance suggest a congenital causation, are met with in cases which are undoubtedly rheumatic.

Dr. Dyce Duckworth has collated the records of 264 cases of mitral stenosis from various sources, including eighty observed by himself, and the figures show that 141, or 60·8 per cent., manifested in some form rheumatic antecedents. Of sixty-four cases observed by myself, and of which I have records, exactly thirty-two (50 per cent.) had been the subjects of rheumatic fever, subacute rheumatism, or rheumatoid pains. The association, therefore, of mitral stenosis with rheumatism is an intimate one. When we come to inquire, however, as to the degree of such association comparatively with that subsisting between mitral regurgitation and rheumatism, I think we shall find the relationship less marked in the one case than in the other, and I hope that the

inquiry will not be unfruitful as regards the determination of the nature of the change which induces mitral stenosis. If I take the cases of mitral regurgitation derived from the same sources (viz., private and hospital practice) from which I obtained the cases of mitral stenosis that I have mentioned, I find that of 123 cases, seventy-three, or 59 per cent., presented evidence of rheumatism in their history. But it must be recollected that in a considerable number of the cases of regurgitation organic disease was not present or not proved, whilst mitral stenosis is always due to organic change. It follows that the figures do not sufficiently express the relation between rheumatism and the organic change which induces *regurgitation* at the mitral orifice. From the analysis of cases of cardiac disease prepared from the records of the London Hospital by Dr. Gabbett for the year 1880, it will be found that whilst 58 per cent. only of the cases of mitral stenosis presented a history of rheumatism, 77 per cent. of the cases of mitral regurgitation were rheumatic.

And now, to push this question further, let us inquire as to the *degree* of manifestation of rheumatism in the two classes of cases. First, as to the relation with acute rheumatism. If we examine the records of acute rheumatism in the London Hospital for 1880 and 1881 we find that the proportions of cases of mitral stenosis (including those in which stenosis was combined with regurgitation) stand thus:—Proportion to all cases in a first attack of acute rheumatism, 5·6 per cent.; in patients suffering a second attack, 3 per cent.; in those with a history of two or more previous attacks, 1·7 per cent. It is obvious that this relationship differs very widely from that existing between mitral *regurgitation* and acute rheumatism, where the proclivity to the lesion increases with the attacks. It is obvious, therefore, that a close relation does not obtain between mitral stenosis and the acute forms

of rheumatism, and that repeated attacks do *not* generally tend to produce the lesion.

As a further step towards the elucidation of the question, I will now ask you to follow me in the inquiry as to the etiology of mitral stenosis in the cases of children. I think you will agree with me that a considerable light can be thrown on the subject from this source. Contradictory opinions have been enunciated as to the proclivity of children to the affection. Dr. Hayden thought that it was to be met with most frequently in children; while Dr. Fagge had no patients under ten, and the youngest observed by Dr. Dyce Duckworth was fourteen. The cases I shall now call your attention to were all under twelve years of age; I have had many who were seven years old. I have tabulated these cases according to the degree of manifestation of rheumatic symptoms. In those who suffered from *acute rheumatism* I found twenty-four cases of mitral regurgitation to one of mitral stenosis; in those classed as *subacute rheumatism*, thirteen of mitral regurgitation to two of mitral stenosis; in those who suffered only *rheumatoid pains*, six of mitral regurgitation to two of mitral stenosis. So far as this evidence goes, therefore, it tends to show that it is not the more severe, but the slighter forms of articular rheumatism, which are attended with the obstructive lesion, whilst the opposite is the case as regards the regurgitant.

To pursue the point, where the rheumatic tendency is not so obvious, but where, as I have said in my first lecture, a rheumatic form of endocarditis is nevertheless manifest, we will consider the cases occurring after scarlatina and measles. In cases presenting a history of scarlatina I found thirteen cases of mitral regurgitation to two of mitral stenosis; in those with a history of measles, twelve of mitral regurgitation to two of stenosis. Lastly, in the case of children

in whom no history of rheumatism was manifest, nor any disease which we might suppose to be likely to induce endocarditis, in these I found twenty-four cases of mitral regurgitation to *fourteen* of mitral stenosis. It is obvious, therefore, that the proclivity to the obstructive lesion is in a very marked manner greatest where articular phenomena are not manifest at all. It might be thought that this was evidence rather against the view that rheumatism is a cause of mitral stenosis; but, as I have shown in my former lecture, the advent of endocarditis having the essential characters of that associated with rheumatism may be so insidious that no subjective sign marks its onset, and we have found in many instances that the course of the affection in the non-articular examples and the morbid changes, as shown by post-mortem examination in the fatal cases, do not differ in any appreciable way from those which are manifest in cases having a distinct history of rheumatic causation. It would, therefore, appear most probable that the correct conclusion is not that mitral stenosis is independent of rheumatism, but that it is associated with the less pronounced forms of it—with its insidious, and not, so to speak, with its *explosive* varieties.

And now let us consider the evidence which clinical observation affords us of the mode of onset of the obstructive mitral lesion. I will give, as briefly as possible, some cases illustrative of the various ways in which the clinical signs indicate the disease to arise.

I. *Presystolic murmur developing insidiously without signs of Rheumatism.*—A lady (Mrs. M.), aged fifty-two, came under my care in 1876 for dyspepsia with very slight jaundice. She manifested no history of, nor predisposition to, rheumatism. I had frequent opportunities of examining the heart, and there were no signs whatever of lesion. In

January, 1877, there having been no symptoms other than an occasional slight dyspepsia previously, the patient complained of "fluttering at the heart," and I found just right of the apex a rough presystolic murmur abruptly terminated by the impulse. I do not think it possible that such sign could have been overlooked in my previous examinations. I can have no doubt that the lesion of stenosis developed gradually without any subjective signs to mark its onset. I have watched the case at intervals ever since: there have been no articular phenomena. The presystolic murmur has been attended with quasi-reduplication of the second sound, and a few months after its first becoming evident a short systolic murmur at the apex was observed also. The systolic murmur increased in intensity, the presystolic continuing to be entirely characteristic. During the whole period until the present there have been no articular troubles, and the cardiac complication, though giving rise occasionally to very slight symptoms, is for the most part, and for long periods, accompanied by no signs of discomfort. This case affords evidence that, in adults, the morbid change can occur in a gradual and insidious manner, with no rheumatic nor other notable phenomena to mark its onset and progress. I have previously given many illustrations to show that a similar course is often manifested in the cases of children who come under treatment for the *consequences* of the cardiac lesion which has been so insidiously effected.*

II. *Systolic murmur at apex becoming changed to presystolic murmur.*—The following notes are condensed from a report by my former house-physician, Dr. J. Needham, by whom the case was carefully watched. John W. D., aged eighteen, was admitted under my care at the London

* "Clinical Lectures on Diseases of the Heart in Childhood," *Medical Times and Gazette*, December 27, 1879, page 711.

Hospital on October 17, 1877. Patient had had so little subjective symptoms that he said that, with the exception of chicken-pox, he had never been ill in his life until eight weeks ago. He had, however, been under treatment for *psoriasis* at intervals for nine years. His present illness was attended with pains in the limbs and abdomen. There was no effusion into the joints, and the temperature never rose above 100·2° Fahr. On admission a soft systolic murmur was noted in the mitral area, the outline of the heart, as determined by percussion, not differing from the normal. Two days after admission the systolic murmur was described as loud and conducted towards left axilla. Seven days after admission there was slight thrill at apex. Fifteen days after admission, the note says: "The cardiac conditions are considerably altered. There is now a well-marked thrill at apex, and, instead of the systolic murmur, there is a well-marked harsh murmur, increasing in intensity and terminated by a clear first sound. About two inches nearer the sternum a blowing systolic murmur is distinctly audible." The systolic murmur (which was in the tricuspid area) subsequently disappeared, and the presystolic became louder, terminating with a sudden uncomplicated first sound. The patient improved under treatment, but suddenly, six weeks after admission, became epileptic.

In this case there was no history of acute rheumatism, though probably the psoriasis was an indication of a rheumatic tendency. In other cases we have distinct evidence that the murmur of mitral regurgitation developed in relation with acute rheumatism may be, in course of time, accompanied by the murmur of mitral stenosis. We may take, as an example, the case of Lydia Grace P., a child of eight, admitted under my care at the North-Eastern Hospital in 1872. She suffered from acute rheumatism. Whilst

under observation a systolic murmur developed at the apex. She was discharged convalescent, and re-admitted in January of the following year with a second attack of rheumatic fever. There was now evidence of mitral regurgitation, with cardiac hypertrophy. She was again discharged convalescent, and re-admitted on August 13, 1873, with a third attack of acute rheumatism. She now manifested well-marked *presystolic*, as well as systolic, murmurs at the apex. She was again discharged convalescent. I do not think it necessary to multiply examples—I have observed many such—of this mode of induction of the condition of mitral stenosis. I may add, however, that it would appear that in some cases the condition of regurgitation is *replaced* by that of stenosis. For example, in a child of nine (Elizabeth M.), a systolic bruit in July, 1869, was found to be accompanied by a presystolic in November; and two years afterwards a presystolic alone was audible, terminated by a sharp and loud impulse.

III. *A presystolic murmur developing insidiously may subsequently be found to be accompanied by a systolic murmur.*—Arthur V., aged eight, was admitted under my care at the North-Eastern Hospital for Children on December 30, 1874. He had never suffered from any definite disease, save measles and whooping-cough at three years of age, but he had frequently been ailing. He manifested a highly pronounced presystolic thrill at the apex, and the presystolic impulse of the left auricle was easily demonstrated on the surface of the chest-wall. A well-marked presystolic murmur was abruptly terminated by the impulse of the ventricle. There was evidence of enlargement of the right chambers, but not of the left ventricle. On January 5 of the following year symptoms of subacute rheumatism became manifest, and then a systolic murmur became evi-

dent at the apex. Subsequently the systolic murmur increased in loudness, and was heard over a wide area, whilst the presystolic was only audible at a point just below and internal to the left nipple. Signs of want of compensation now became more marked, and œdema—which, however, disappeared under treatment—supervened. Such a history is by no means uncommon; the signs of regurgitation supervene on those of stenosis, and the double lesion becomes manifest.

I hope that the evidence which I have brought forward may enable us to see in a clearer light the mode of development of mitral stenosis. This evidence, as I consider, tends to show that in a considerable number of cases the origin and course are insidious and gradual. The disease is not independent of rheumatism, but is often unaccompanied by pronounced rheumatic phenomena; it is initiated by the form of endocarditis which I sketched in my first lecture as manifested by no subjective sign, accompanied by no prominent symptom, and yet differing in no essential feature from that which occurs in obvious relation with rheumatism. The endocarditis which results in mitral regurgitation is more violent, so to speak, whilst that which initiates stenosis is more protracted, giving rise to a slower formation of fibrous, quasi-cicatricial tissue that under the even pressure of blood in the auricle tends to form the smooth septum which has erroneously suggested a possible congenital causation.

Not all the cases of mitral stenosis, however, originate in this manner. In some there has been first the induction, in association with the phenomena of acute rheumatism, of the lesion of mitral regurgitation; then has occurred probably a slow welding of the curtains; and in the repeated attacks of endocarditis the changes have been slower than

those which result in retraction of curtains, cords, and columns to the ventricular wall.

By either of these modes produced, it is probable that secondary changes take place in the diseased tissue—under the tension of blood the fibrous septum thickens, for *it* has to bear the chief strain of the auricular pressure, and not the ventricle, as in the case of mitral regurgitation. In some cases it undergoes calcareous degeneration, and probably in others, where gouty signs are manifest, it becomes infiltrated with the earthy lithates.

Compensation in cases of mitral stenosis may be maintained, as in mitral regurgitation, for long periods. It may be even more simple in the former case than in the latter, for it is only a hypertrophy of the right ventricle, and not of both ventricles, that is needed to sustain it. The left ventricle, not being dilated, continues to afford a sufficient *point d'appui*, and it only needs the *vis à tergo* of a strong right ventricle, aided by a hypertrophied (or at least not enfeebled) auricle, to urge a sufficiency of blood through the narrowed orifice. So long, therefore, as a good nutrition maintains the muscular power of right ventricle and left auricle, any special methods of treatment of a simple condition of mitral stenosis may be unnecessary. In course of time, however, the right ventricle or left auricle, or both, may begin to fail. Usually it is the former, but I have quoted a case in which it was markedly the latter, and in this I have no doubt the muscle failed on account of the great privations which the patient had undergone. The right chambers dilate on account of the pressure which is maintained within them if the compensatory muscular power begins to fail. Then ensue the dyspnœa, the œdema, ascites, etc., with which we are familiar in analogous cases of mitral regurgitation. To restore compensation we may use, for the

most part, similar means to those which we have considered in regard to mitral regurgitation. When the gravest troubles of orthopnœa and dropsy have supervened, I have in many cases found that rest, combined with the administration of nutrients and tonics, and with digitalis, have restored the *status quo ut ante*, often for a considerable period.

Coincidently with the use of means for increasing muscular power, I consider that small and repeated abstractions of blood are even more valuable in mitral stenosis than in mitral regurgitation. The tension of the right heart may be sensibly relieved even by a leech or two applied over the præcordium. Dr. Bedford Fenwick has narrated a case which is an amusing as well as instructive example of the value of blood-letting in failure of compensation in mitral stenosis. A patient of Dr. Andrew Clark, at the London Hospital, manifesting the physical signs of mitral stenosis and aortic incompetency, had not improved by a month's treatment with rest ether, senega, and digitalis. There was much dyspnœa, with signs of œdema of the lungs. The urine became scantier, the œdema increased, and coma appeared to be supervening. At this time the patient in his half-consciousness struck his own nose and brought on a copious epistaxis. Shortly after, consciousness returned, a copious diuresis followed during the night, and in less than a week the œdema disappeared, and the patient became convalescent. I quite agree with Dr. Bedford Fenwick that abstraction of blood by leeches or cupping is too much neglected in the cases we are considering, and that it is to be justified both by theory and practice.*

As regards the special action of *digitalis* in restoring compensation in cases of mitral stenosis, I am not convinced that

* "On the Use of Venesection in Cases of Heart Disease," by Bedford Fenwick, M.D., M.R.C.P.: *Lancet*, August 5, 1882, page 179.

this is so markedly proved to be beneficial as in the cases of mitral regurgitation. I have found that in some instances, as shown by the sphygmograph, digitalis has restored regularity, whilst in others it has increased irregularity of pulse. I believe it to be most valuable where stenosis and regurgitation are combined. Where the right ventricle is chiefly at fault I do not think its good effect is so manifest; where it can induce an efficient systole of both ventricles and co-ordinate them, then I think it is the more valuable. In failure of the right heart, therefore, in extreme mitral stenosis I look more hopefully to caffeine and to *Convallaria maïalis*.*

M. Sée has narrated three cases of mitral stenosis in which the extract of convallaria was administered. In the first there was a marked diuretic effect, the quantity of urine increasing under treatment from an average of one litre to two litres and a half and three litres, together with a great amelioration of the dyspnœa which was manifest on exertion. In the second case, evidencing œdema, ascites, and grave signs of cardiac failure, after a dose of one gramme per diem of extract of convallaria, marked diuresis occurred, and œdema disappeared in two days. Oliguria returned, and the dose was increased to one gramme and a half. In successive days the quantity of urine passed increased in the following proportions:—600 grammes, 2200 grammes, 2400 grammes, and 3000 grammes; it then fell to 2000 grammes, all signs of œdema and ascites having disappeared. The third case was one in which diabetes mellitus co-existed with mitral stenosis. In this case a very marked amelioration of the symptoms of imperfect compensation is recorded.

* I have to thank Messrs. Savory and Moore, and Mr. Brownen, F.C.S., for exhibiting not only the various preparations of convallaria, but the alkaloids (convallarin and convallamarin) obtained from the plant.

Considering the absence of violence in the storm which, involving the endocardium, leaves behind it the condition of obstruction, we may ask whether the result, stenosis, is not more innocent than the result of the more violent storm, regurgitation? The question is a difficult one. We can point to many cases of regurgitation where there has been an arrest of all morbid process, and where fair health has been maintained for long periods of years. Such instances are, I think, less common in mitral stenosis—there is not a like quiescence; and degenerative changes or intercurrent morbid phenomena are more likely to occur. The average age at death in nineteen cases of mitral stenosis observed by myself was thirty-five years. In forty-two fatal cases collected by the late Dr. Hayden it was 37·82 years.

In the cases both of mitral stenosis and of mitral regurgitation, however, it is not alone with the simple dynamical problem of the restoration of muscular compensation that we have to deal. In every case we have to weigh the probability of complications arising—complications so intimately associated with the conditions as to present an essential matter for consideration in any question as to treatment. Such are (1) repeated attacks of pericarditis or endocarditis, and (2) embolism.

A patient who has once suffered from rheumatic affection of the endocardium is liable, of course, to a repetition of the morbid process. With such pericarditis is by no means infrequently associated. In children I am strongly of opinion that pericarditis, when resulting in adhesion of the two layers of the pericardium (and often accompanied with fibrous proliferation amongst the muscular fibrils), is a grave cause of danger, spoiling the chance of compensation, and greatly interfering with the beneficial results of treatment. In cases in young people, where compensation fails even

under suitable treatment and good nutrition, where evidences of cardiac hypertrophy and dilatation are in excess of those which usually accompany the valvular lesion, we may, I think, generally conclude that the pericardium is adherent.

An accident of this condition both of regurgitation and stenosis (especially the latter), yet intimately connected with them, is the occurrence of *embolism*. The consideration of this is often forced upon us when the question occurs as to treatment.

Let us consider this matter from its *clinical* aspect.

I. A case presents itself to us with symptoms of cough and dyspnœa. The onset of these symptoms has been sudden, perhaps initiated by a rigor. There may have been slight pyrexia or none. The characteristic feature is that, after a short time, when the cough has been attended with mucous and frothy sputa, the expectoration is observed to be coloured with bright blood. In many such instances we may find localised patches of dulness in the upper or the lower thoracic regions, with a few muco-crepitant râles; in others neither dulness nor moist sounds can be detected. We examine the heart, and find evidence of mitral stenosis or (with less probability) mitral regurgitation. The existence of these signs, especially when the lesion is in the upper lobes, may cause us to fear the advent of pulmonary phthisis; but observation shows us that, though the hæmoptysis may occur again and again, the changes of tubercle are not manifest. Or whilst a case is under treatment for the symptoms of ill-compensated stenosis or regurgitation, a sudden attack occurs of dyspnœa with physical signs of a localised broncho-pneumonia. In some cases the outline of dulness can be delineated as a defined triangle. It is broncho-pneumonia differing from the ordinary form, for its

origin may be entirely independent of catarrhal influences, and it has a special feature—the occurrence of hæmoptysis. It is rarely that a case of mitral stenosis goes through its course without the manifestation of some such phenomena. Dr. Hayden records that hæmoptysis was noted as a symptom in forty-four cases out of eighty-one of mitral stenosis (54·3 per cent.). The history of fatal cases generally shows the repeated development of such areas of condensed lung. Morbid anatomy affords the clue to the interpretation of these phenomena. In many instances the right auricle is found to contain, adherent to its *musculi pectinati*, fibrinous coagula, and detached masses from these have been found to block branches of the pulmonary artery. From such infarctions result the appearances formerly described as "pulmonary apoplexy."* The infarct may in many cases be undiscoverable, for the plug undergoes fatty degeneration and solution, and the lung-tissue may present no naked-eye changes. I am inclined to think, however, that what is true of the grosser is true of the finer changes, and that the hæmoptysis or the limited broncho-pneumonia of mitral stenosis is due to plugging (it may be of small twigs) of branches of the pulmonary artery.

Now as regards treatment when such phenomena are manifest. In the first class of cases, where no sign of ill-health has previously been prominent, I would accept the occurrence as evidence that compensation is disturbed. There is an abnormal retardation of the circulation in the right chambers of the heart, and we are called upon to use some of the means we have described for increasing the power of the ventricles. In all cases it will be advantageous

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* Embolism of the pulmonary artery was found in eleven of sixty eight fatal cases of cardiac disease of which I have records.